يمنع التعامل على هذا الملف الإ
بعد الرجوع الإ الرعاة الرسميين
ولكن تم إعداده خوفا من ضياع
محتوياته

نهاية العالم
نبوة نبي

FACULTY OF VETERINARY MEDICINE BENH UNIVERSITY

ZOONOSES-3014-

COURSE NUMBER 8228

EDITED BY STAFF MEMBERS OF ZOONOSES

فاتحة-إستهلال-مقدمة-ديباجة-مطلع-توطئة-تمهيد-تصدير

PREFACE

PREFACE

1-ZOONOSES ARE CONSIDERED TWO GROUPS OF COMMUNICABLE DISEASES

2-THOSE TRANSMITTED FROM VERTEBRATE ANIMALS TO MAN WHICH ARE STRICTLY SPEAKING ZOONOSES (FIRST GROUP)

3- THOSE COMMON TO MAN AND ANIMALS (SECOND GROUP)

4-THE FIRST GROUP ,THE ANIMALS PLAYING AN ESSENTIAL ROLE IN MAINTAINING THE INFECTIONS IN THE NATURE AND MAN IS ONLY AN ACCIDENTAL HOST

5- IN THE SECOND GROUP BOTH ANIMALS AND MAN GENERALLY CONTRACT THE INFECTIONS FROM THE SAME SOURCES SUCH AS SOIL ,WATER , INVERTEBRATE ANIMALS AND PALNTS AS ARULE ,HOW EVER ANIMALS
DO NOT PLAY AN ESSENTIAL AROLE IN THE LIFE CYCLE OF THE ETIOLOGIC AGENTS,,
BUT MAY CONTRIBUTE IN VARYING DEGREES TO THE DISTRIBUTIONS AND ACTUAL TRANSMISSIONS OF INFECTIONS.
6-NO ATTEMPT HAS BEEN MADE TO INCLUDE ALL INFECTIONS AND DISEASES COMPRISED IN THESE TWO GROUPS
7-ASELECTION HAS BEEN MADE OF SOME THAT ARE OF PRINCIPLE INTERESTS FOR VARIOUS REASONS IN THE FIELD OF PUBLIC HEALTH

8-THE NUMBER OF LISTED ZOONOSES IS ABOUT 176 DISEASES AND INCREASED AS ANEW BIOMEDICAL KNOWLEDGES ARE ACQUIRED

9-MORE OVER AS HUMAN ACTIVITY EXTENDS INTO UN EXPLORED TERRITORIES CONTAINING NATURAL FOCIS OF INFECTIONS

10-NEW ZOONOTIC DISEASES ARE CONTIUALLY BEING RECOGNIZED

11- AS BETTER HEALTH DIFFERENTIAL DIAGNOSTIC METHODS IMPROVED HAVE DISTINGUISHED ZOONOSES PREVIOUSLY CONFUSED WIYH OTHER MORE COMMON DISEASES.

12- AS ARULE ZOONOSES ARE TRANSIMITED FROM ANIMALS TO MAN BUT THE REVERSE MAY ALSO OCCUR ,THE VICTUMS IN THESE CASES ARE ANIMALS WHICH MAY IN TURN RETRANSMITT THE INFECTIONS TO MAN UNDER CIRCUMSTANCES .

13-ZOONOSES HAVE APROFOUND INFLUENCES ON PUBLIC HEALTH AND THE LIVE STOCK ECONOMYIES OF DIFFERENT REGIONS OF THE WORLD SO IT LIES ON THE VETERINARIANS TO PROTECT HUMANITY FROM THESE ZONOOSES .

14- THEREFORE, THE VETERINARY PROFESSION IS NOT ONLY OF AN ECONOMIC IMPORTANCE BUT ALSO OF CONSIDERABLE SANITARY

متبحر –عويص-عميق-بالغ-بليغ-شامل-سحيق-كامل-لجى-

IMPORTANCE TO ENVIRONMENTAL PUBLIC HEALTH.

ZONOOSES

Section number one WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD

MOHAMMED YOUSEF WEHABA

Definitions and classification of zoonosis

1-it is agreed that the zoonoses of exact biological merit

إستحقاق

2-it were creates common ground for the medical and veterinary professions to explore the epidemiology of diseases that man acquires from both domestic and wild animals and advised techniques for their control.

explore -يستكشف –إستجلى-إستطلع-إستعرض-إستقرأ-إستقصى-إستكشف-إستوضح-

Epidemiology -الوبائيات التحليلية –علم الأمراض الوبائية-علم وبائية المرض-علم الأوبئة-دراسة وبائية ويصف الرابطة بين نظرا للسلوكيات أو الظروف البيئية والصحة البدنية أو النفسية عن طريق رصد الظواهرفى العالم الحقيقى من خلال البيانات الإحصائية . باللغة اللاتينية epiبمعنى على أو بين demos بمعنى الناس logosبمعنى الدراسة – دراسة أنماط وأسباب وأثار الصحة وظروف المرض وهذا يعنى أن دراسة كلمة علم الأوبئة لها جذورها فى دراسة ما يصيب السكان .هودراسةالتوزيع ومحددات الأحوال ذات الصلة بالصحة أو الأحداث المحددة فى مجموعة سكانية وتطبيق هذه الدراسة بغرض السيطرة على المشاكل الصحية . وتصف الدراسات من الرصدية إلى التجريبية .ويقال أن أبقراط هو أب الطب .

3-previously zoonoses were defined as those diseases and infections which are naturally transmitted between vertebrates animals and human.

Zoonoses - الأمراض حيوانية المنشأ-الأمراض حيوانية المصدر-الأمراض الحيوانية المصدر

4-it has been argued that this definition is too wide in that it includes not only infections

Argued جادل-موضع جدال-يتجادل-حاورجدلا-تجادل-حوجة-غيرمتفق-يدلل-يناقش-ترافع-تشاد-علل-باحث-بحث-حاج-مارى-نازع-إختلف-إختصم-تباحث-تحاور-تحاجج-تشاجر-تخاصم

acquires from animals but also includes :

acquire - يكتسب –مكسوب-ممتلك-إستجلب-إستولى على –اجلب-أحرز-أدرك-أصاب-أفاد-أمن-أنال

a-diseases produced by non-infective agents such as toxins and poisons

toxins–سموم-ذيفانات-سم قاتل-المادة السامة

b-infections that animals acquires from man that are merely incidental infections of no public importance .

merely --مجرد-لا غير- ليس إلا – ليس إلا – ليس سوى –جرد-فحسب-لايخرج عن-لا يعدو كونه-بركة حد-قسيم-لاحقة-معلم تحديد-بحيرة-تخم-جزء-مجرد-((ويقال لا شئ بعدذلك))

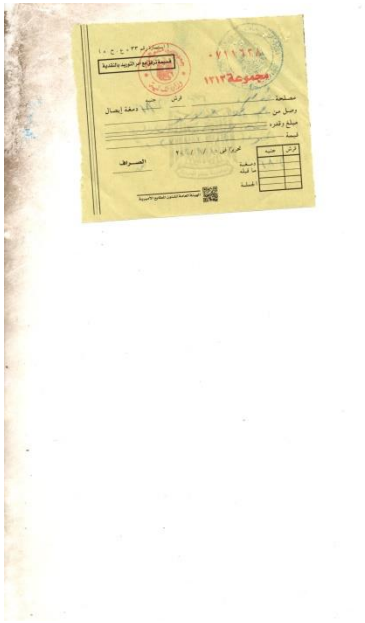
JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
28109091800516 - 2004578801966

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA



مجموعة ١٢١٣

درجة دوكتوراة البكتريا والمناعة والفطريات -الساعات المعتمدة-نيرم اول -رقم جلوس اختبارى ٣٠١٤

كود المقرر COURSES CODES NUMBERS	المقرر الدراسى UNIVERSAL STUDYING HOURS NOMINATIONS	الدرجات الحاصل عليها ٣٠١٤ EXAMS INCOMES DEGREES	التقدير RANKS	الرقمى APPOINTMENTS
8051 ٨٠٥١	الوراثة البكتيرية BACTERIAL GENTICS	٩٣	A	A
8057 ٨٠٥٧	الكتريا المسببة للتسمم الغذائى FOOD BORNE POISONING MICRO-ORGANISMS	٩٤	A	
8142 ٨١٤٢	التقنيات البيولوجية والاجهزة MOLECULAR DIGNOOSTICS DEVICES	٨٢	B	
8053 ٨٠٥٣	سيمنارات فى مجال التخصص LECTURERS	٤٧	A	

مركز البحوث الزراعية
معهد بحوث صحة الحيوان
قسم الطفيليات الإدارية

تحريرا في: ٢٠٢٢/١١/٢
Time:11:21:07

بيان حالة شخصي

الرقم القومي: ٥٩٠٠ — الرقم التأميني: ٥٩٠٠

الاسم: محمد محمود محمد يوسف وهبه

تاريخ الميلاد: ١٩٨١/٠٩/٠٩ — محل الميلاد: ابو حمص

الحالة الإجتماعية: اعزب — النوع: ذكر

المؤهل — تاريخ وجهة الحصول عليه

١-بكالوريوس طب بيطري — ٢٠٠٣ — ج.التيسير
٢-ماجستير تخصص الميكروبيولوجيا والمناعه — ٢٠٠٩ — ج.النشيخ
٣-دكتوراه تخصص الميكروبيولوجيا والمناعه
٤-
٥-

الوظيفة: مساعد باحث — المرتب الأساسي: --------

الدرجة: مساعد باحث — تاريخ الحصول عليها: ٢٠٢٠/٦/٩

تاريخ التعيين: ٢٠١٢/٠١/٢٢ — تاريخ استلام العمل: ٢٠١٢/٠٣/٠٥

جهة التعيين: المعهد (زراعة القاهرة الجديدة) — رقم قرار الدرجة: ٢٠٢٠/٢١٤٣

التعيين: ٢٠٠٨/٠٨/١٧ ق.ر.٢٠١٥/٣٠٤٠ — القسم/المعمل: م/طفا

تاريخ الإحالة للمعاش: ٢٠٤١/٠٩/٠٩

تقارير الكفاية: يعامل بقانون التأمينات رقم٧٩لسنة١٩٧٥

الحاسب الآلي — الموظف المختص — رئيس القسم — مدير الإدارة العام

بيانات خاصة

محل الإقامة: ق العمرو - كفر الزيات مركز كفر الزيات الغربيه
رقم البطاقة: ١٨٠٠٥١٦ — تاريخ صدورها: ٢٠١٨/٠٧
قسم/مركز: الغربيه — الموظف من النخبة: أدى

Arab Republic of Egypt
Ministry of Agriculture
Agricultural Research Center
Animal Health Research Institute

السيد الأستاذ الدكتور / عميد كلية الطب البيطرى
جامعة بنها
تحية طيبة وبعد...

تتشرف بالإفادة بأن المعهد ليس لديه مانع من قيد طالب / محمد محمود محمد يوسف وهبه ضمن المقيدين للدراسات العليا (الدكتوراه) (بقسم البكتريولوجى) بالكلية " إدارياً فقط" وذلك لحين تحديد موضوع الدراسة من خلال ثلاث مواضيع تحددها لجنة الأفكار البحثية بالمعهد للعرض على الأستاذ الدكتور مشرف الكلية لتحديد موضوع الدراسة ومن ثم العرض على اللجنة البحثية المختصة بالمعهد للمناقشة والرأى فى ألفاق موضوع الدراسة مع الخطه البحثيه للمعهد.

ملحوظه:" هذه الموافقه لاتلزم المعهد بأى مستحقات ماليه أو إداريه (لقرع الدارسه) مالم يتم استكمال اجراءات التسجيل بالمعهد ومن ثم موافقة مركز البحوث الزراعيه.

رجاء التفضل بإطلاع والتكرم بالاحاطه.

وتفضلوا سيادتكم بقبول فائق الاحترام ...

تحريرا ٢٠٢٢/٨/١٤ م

وكيل المعهد للبحوث وخدمة البيئة
أ.د/ هشام على فهمى

مدير المعهد
أ.د/ ممتاز محمد الهدى شاهين

العنوان : ٧ شارع ناصح العبيد - الدقى - الجيزة - ص. ب : ١٣١١٨ تليفون : ٣٧٤٨٩٣٣٠ - ٣٧٤٨٩٠٢٢ فاكس : ٣٧٥٥٠٠٣٠
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Web Site : www.shrl.gov.eg E.mail : Ahri@arc.sci.eg

إستماره تسجيل

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نوع تحقيق الشخصية : رقم قومي رقم تحقيق الشخصية : 28109091800516
وظيفة الطالب : GOVERNMENT/ASSISTANT RESEARCHER

العنوان الدائم

رقم المنزل : 3 اسم الشارع : جامع السلام المحافظه : الغربية

العنوان الحالي

رقم المنزل : 3 اسم الشارع : جامع السلام المحافظه : مصر

وسائل الاتصال

وسيلة الاتصال : التليفون البيان :

بيانات المؤهل

الدولة : مصر الجامعة : جامعة بنهـــا الكليـــة : كلية الطب البيطري
مستوى المؤهل : دراسات عليا المؤهل : ماجستير الميكروبيولوجيا والفطريات والمناعه شعبة المؤهل : البكتريولوجيا
سنة المؤهل : 2022-2021 تاريخ الحصول على المؤهل :

الدرجة العلمية المسجل عليها

الكليـــة : كلية الطب البيطري الدرجة العلمية : درجة دكتوراه الفلسفة فى العلوم الطبية البيطرية
القسم والشعبة : قسم البكتريولوجيا والفطريات والمناعة
الشهادة المسجوعه : دكتوراه البكتريولوجيا والمناعة والفطريات

التوقيع

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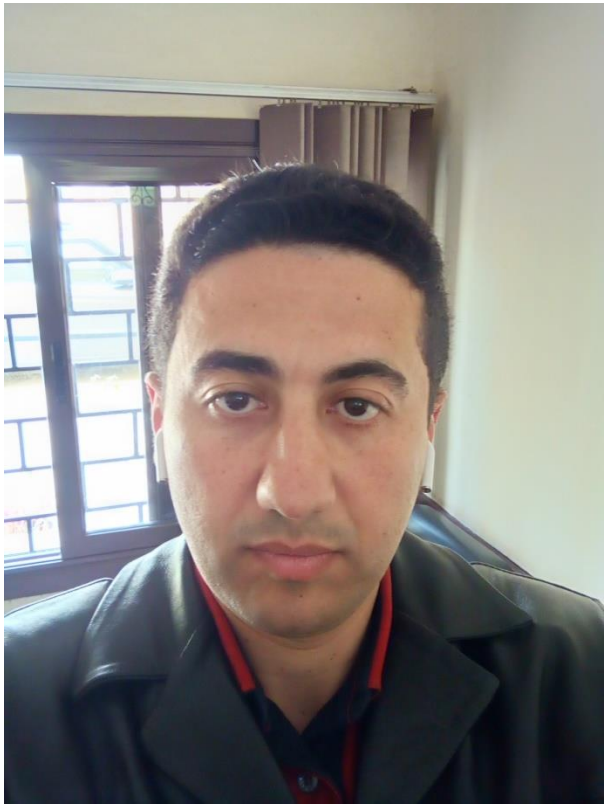

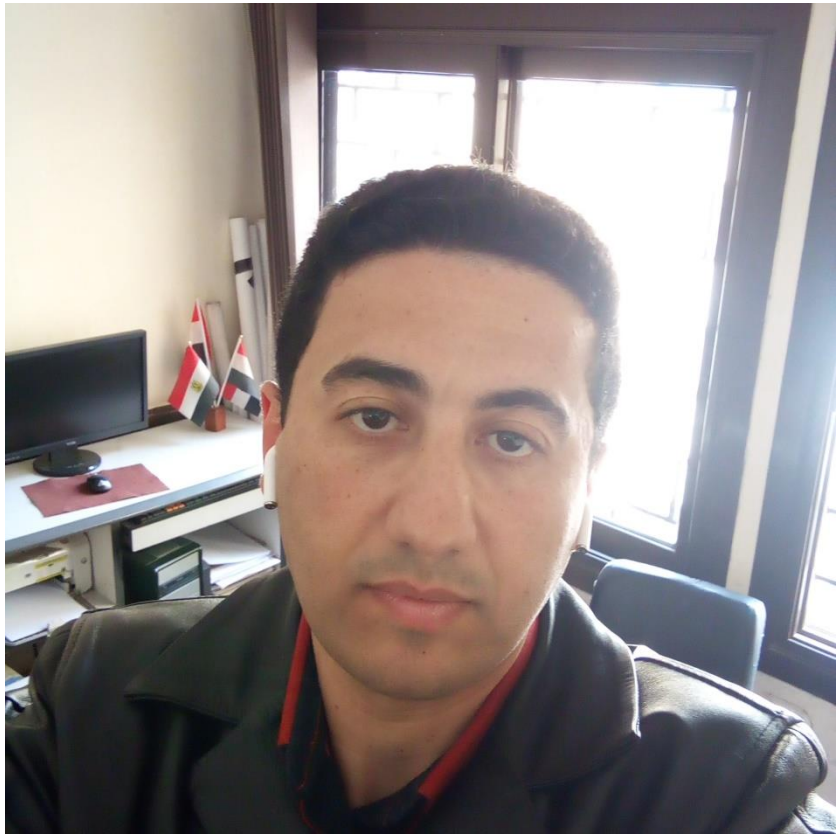

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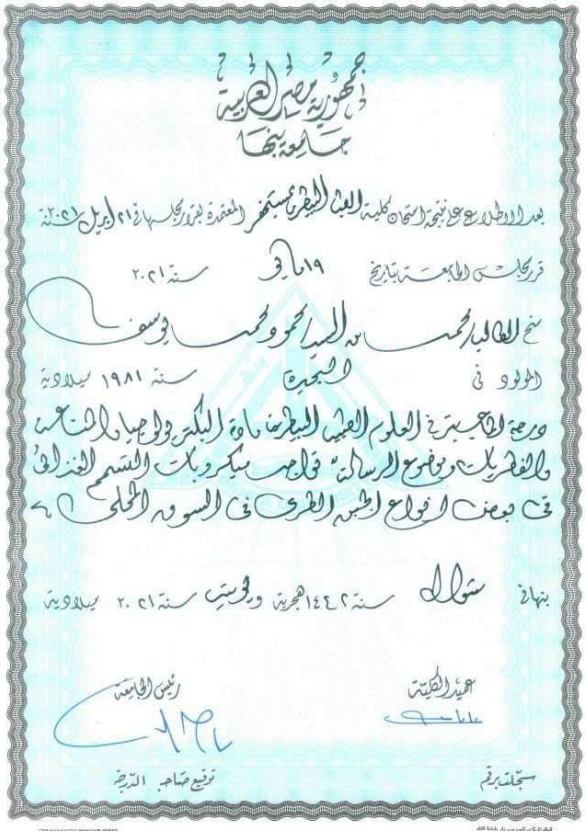
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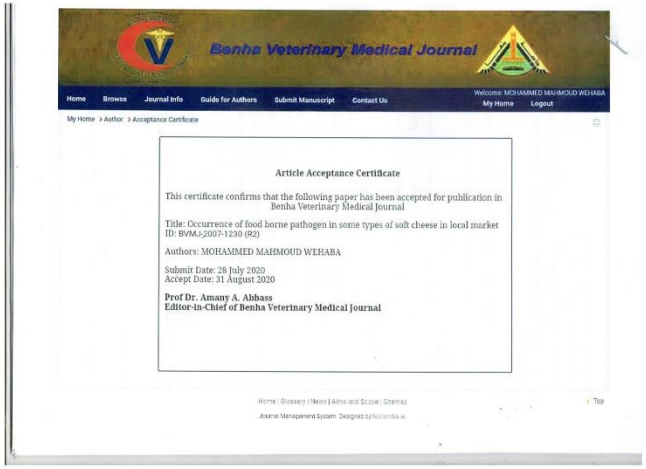
جامعة بنها
بنهاية شوال سنة ١٤٤٢ هجرية وأبريل سنة ٢٠٢١ ميلادية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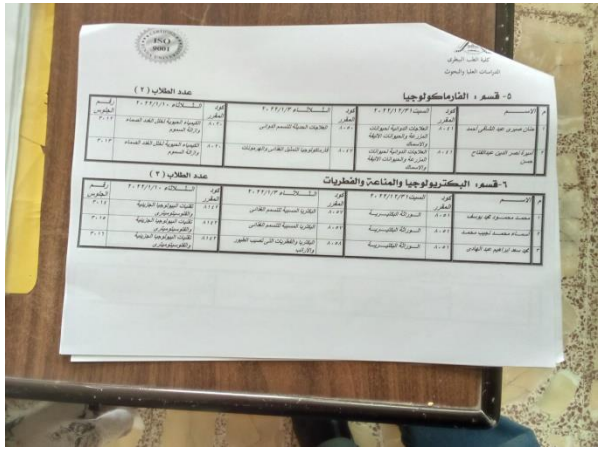
ID: BVMJ-2007-1230 (R2)
Authors: MOHAMMED MAHMOUD WEHABA
Submit Date: 28 July 2020
Accept Date: 31 August 2020
Prof Dr. Amany A. Abbass
Editor-in-Chief of Benha Veterinary Medical Journal



كلية الطب البيطري
الدراسات العليا والبحوث

٥- قسم : الفارماكولوجيا

عدد الطلاب (٢)

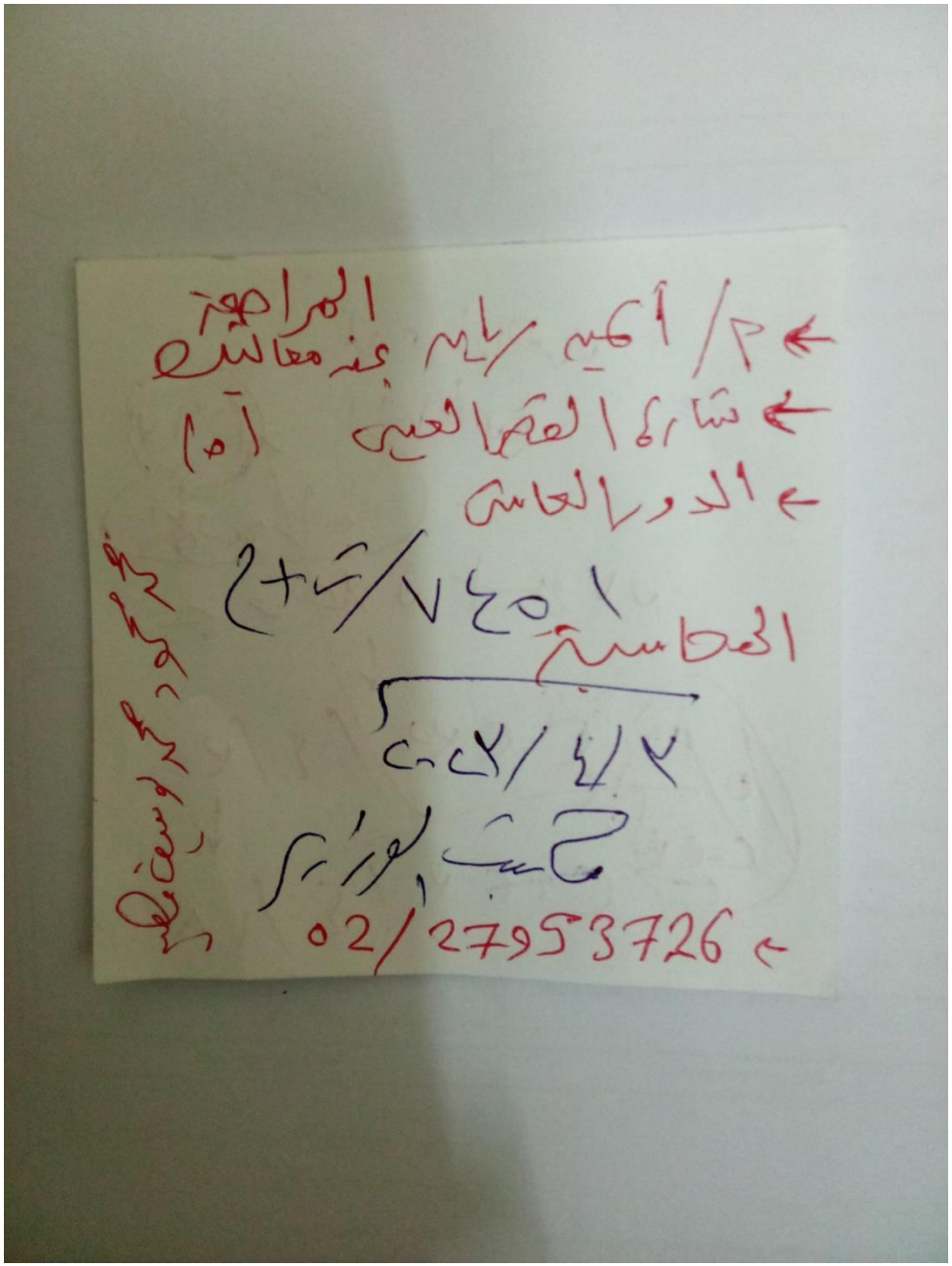
م	الاسم	الفرقة / المقرر	السبت ٢٠٢٢/١٢/٣ الساعة ١٠	الفرقة / المقرر	الساعة ٣ ٢٠٢٢/١٢/٣	الفرقة / المقرر	الثلاثاء ٢٠٢٢/١٢/٦ الساعة ١٠	رقم الجلوس
١	عثمان صبري عبد الشافي أحمد	٥٠٤٠	الجلوكوما	٥٠٤٠	الجلوكوما	٥٠١٢	الكيمياء الحيوية لطلاب العهد المعداه وزارة السعود	٥٠١٢
٢	أميرة نصر الدين عبدالفتاح الجمل	٥٠٤٠	الجلوكوما	٥٠٤٠	الجلوكوما	٥٠١٣	الكيمياء الحيوية لطلاب العهد المعداه وزارة السعود	٥٠١٣

٦-قسم: البكتريولوجيا والمناعة والفطريات

عدد الطلاب (٢)

م	الاسم	الفرقة / المقرر	السبت ٢٠٢٢/١٢/٣ الساعة ١٠	الفرقة / المقرر	الساعة ٣ ٢٠٢٢/١٢/٣	الفرقة / المقرر	الثلاثاء ٢٠٢٢/١٢/٦ الساعة ١٠	رقم الجلوس
١	محمد محمود كمال يوسف	٥٠٤١	البكتريا	٥٠٤١	البكتريا السببة للتسمم الغذائي	٥٠١٢	الفطريات المجهرية الخزعية	٥٠١٢
٢	إسراء مجدي أنيس محمد	٥٠٤١	البكتريا	٥٠٤١	البكتريا السببة للتسمم الغذائي	٥٠١٣	الفطريات المجهرية الخزعية والقرم سيلو حجرة	٥٠١٣
٣	محمد سعد إبراهيم عبد الهادي	٥٠٤١	البكتريا	٥٠٤٢	البكتريا والفطريات التي تصيب الطيور والأرانب	٥٠١٤	الفطريات المجهرية الخزعية والقرم سيلو حجرة	٥٠١٤

الأنشطة التى يقوم بها المعمل
ahmtanta@yahoo.com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م/ أحمد ربيع
شارع القصر العيني أمام
الدور العاشر
02/27953726

UUWD89J308ULRP4

الشكوى الإدارية فى ملفات الرقابة الإدارية المصرية

BENHA UNIVERSITY

FACULTY OF VETERINARY MEDICINE

DEPARTMENT OF BACTERIOLOGY, IMMUNOLOGY, AND MYCOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Yousef Wehaba

Bachelor of veterinary medicine ,kafer el-shiekh university (2003)

Very Good , The Seventh Rank

Hiring As A Veterinarian Doctor In Animal Health Research Center At (2012)

Master (Bacteriology, Immunology And Mycology, 2021)

Assistant Researcher Animal Health Institute (2020)

Agricultural Research Center,

Ministry Of Agriculture And Land Reclamation

Arab Republic Of Egypt

ترقى ماجيستير.pdf

BACTERIA sSECTION.pdf

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

Bacteriology, Immunology and Mycology department

Third year (post. graduates(

Dr. Manar El-khayat

2021

Family:MYCOPLASMACEAE

Order : MYCOPLASMATALES

family: MYCOPLASMACEAE.

Genus : MYCOPLASMA.

Species:

Mycoplasma bovis

Mycoplasma mycoides.

Mycoplasma var Capri.

Mycoplasma gallisepticum.

Mycoplasma gallinarum.

Mycoplasma hominis.

***Mycoplasma* species are highly fastidious microorganisms characterized by**

- Lacking **cell wall** so it's **pleomorphic in shape**.
- Low G+C content (23%–.(40%
- Small size of genome (0.58-1.4 Mbp.(
- Difficult to culture and slow growing
- Special growth requirements are needed for their growth.
- Grows on solid media giving the characteristic **"fried egg"** colonies when examined under a plate microscope.
- ***M. bovis*** film and spot formation can also be viewed on the surface of solid media which indicates the presence of lipolytic activity.

Morphology and staining characters:

- Individual cells of mycoplasma are extremely pleomorphic ranging from coccoid to coccobacillary to long branching filaments about (100-150 µ.(
- Gram negative
- *Mycoplasma* is easily stained with Giemsa stain

Growth requirement and Cultural characters:

- *Mycoplasma* needs a rich medium containing natural animal protein, a sterol component and moisture is essential.
- Elevated Co_2 and low O_2 are needed and Incubation is best at (33-37°C.(
- of the natural animal proteins the most commonly used is blood serum in a concentration of (10-20%). Serum supplies not only cholesterol but also saturated and unsaturated fatty acids which the organisms are unable to synthesize.
- PPLO media containing 20% horse serum, 10% of a 25% yeast extract، the final concentration of agar is 1%, pH of (7.0-7.8) is advisable.
- Fluid media, the same constituents as solid media but without agar(

On solid media containing 10% bovine serum, *Mycoplasma* produces:

The characteristic microscopic fried egg like colonies

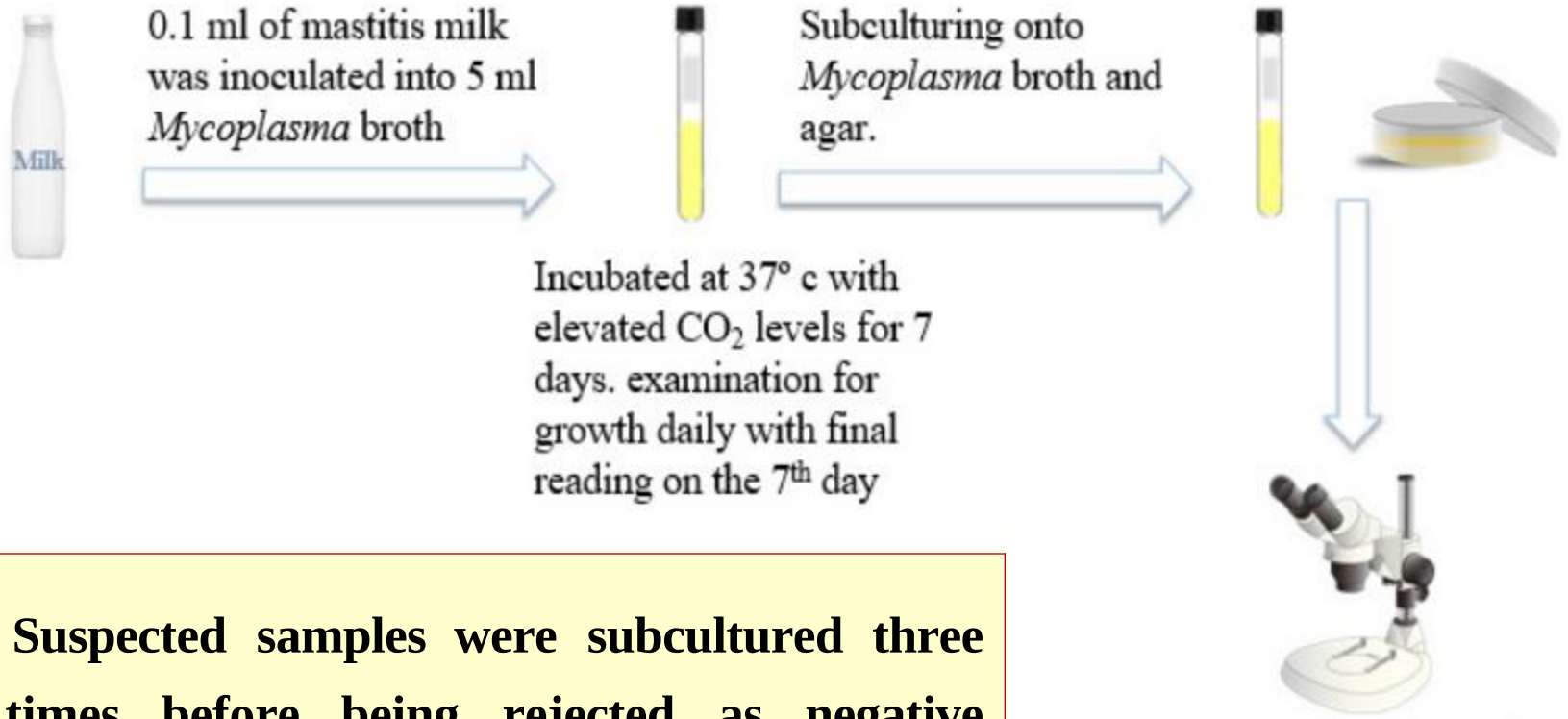
-microscopic small, glistening colonies with an entire edge and a nipple-like darker center and these colonies are usually difficult to remove from the surface of the medium due to the penetration of the organism below the surface of the medium.

For subculturing from agar to broth, agar block with colonies was cut using sterile spatula, and dropped into broth.

In liquid media, *Mycoplasma* produces:

slight turbidity and a finally granular precipitate are formed.

☮ Isolation of *Mycoplasma* from milk samples:


Milk
0.1 ml of mastitis milk was inoculated into 5 ml *Mycoplasma* broth
Incubated at 37° c with elevated CO_2 levels for 7 days. examination for growth daily with final reading on the 7th day
Subculturing onto *Mycoplasma* broth and agar.
Plates are examined using stereomicroscope to detect *Mycoplasma* colonies with the characteristic fried egg appearance.

- ✓ **Suspected samples were subcultured three times before being rejected as negative samples.**
- ✓ **For subculturing from agar to broth, agar block with colonies was cut using sterile spatula, and dropped into broth.**

Biochemical characters:

- Some strains ferment glucose;others do not ferment glucose
- film and spots in egg yolk medium is also helpful.
- *M. pneumoniae* is distinct among humanstrains in its ability to cause complete lysis of mammalian red corpuscles, although most other strains, human and animalstrains produce some degree of haemolysis in blood agar made up with red cells from the guinea pig.
- Argnine hydrolysis test
- Digitonin sensitivity test, Nisin sensitivity tests

By culturing

Acholeplasma spp.
are capable of growing on Hayflick medium

Forming
Mycoplasma-like-colonies

Differentiation between
Mycoplasma spp. and *Acholeplasma* spp.

By different methods as
Digitonin, Nisin sensitivity tests and molecular assays

Accurate diagnosis

Differentiation between *Mycoplasma* species.

Species	Inhibition by digitonin	Glucose	Arginine hydrolysis	Phosphatase	Tetrazolium reduction	Film& spot in egg yolk media	Disease
M.mycoides	+	-	-	-	+	-	Contagious bovine Pleuropneumonia
M.bovirhinis	+	+	-	-	variable	+	Pneumonia in calves
M.bovigentalium	+	-	-	+	Variable	+	Mastitis, vaginitis‹ Arthritis, seminal vesiculitis
M.bovis	+	-	-	+	+	+	Mastitis, arthritis
M.hominis	-	-	+	-	-	-	Pneumonia. Inflammation of Urogenital tract

Antigenic characters and Serological behavior:

Mycoplasmas are:

- separated into definite number of serotypes.
- Certain species possesses haemagglutinating, haemadsorbing and cell attaching properties.
- In being inhibited or neutralized by specific antiserum.

The common methods used for studying the antigenic structure are:

-Growth inhibition

-Gel-diffusion test.

-Complement fixation test

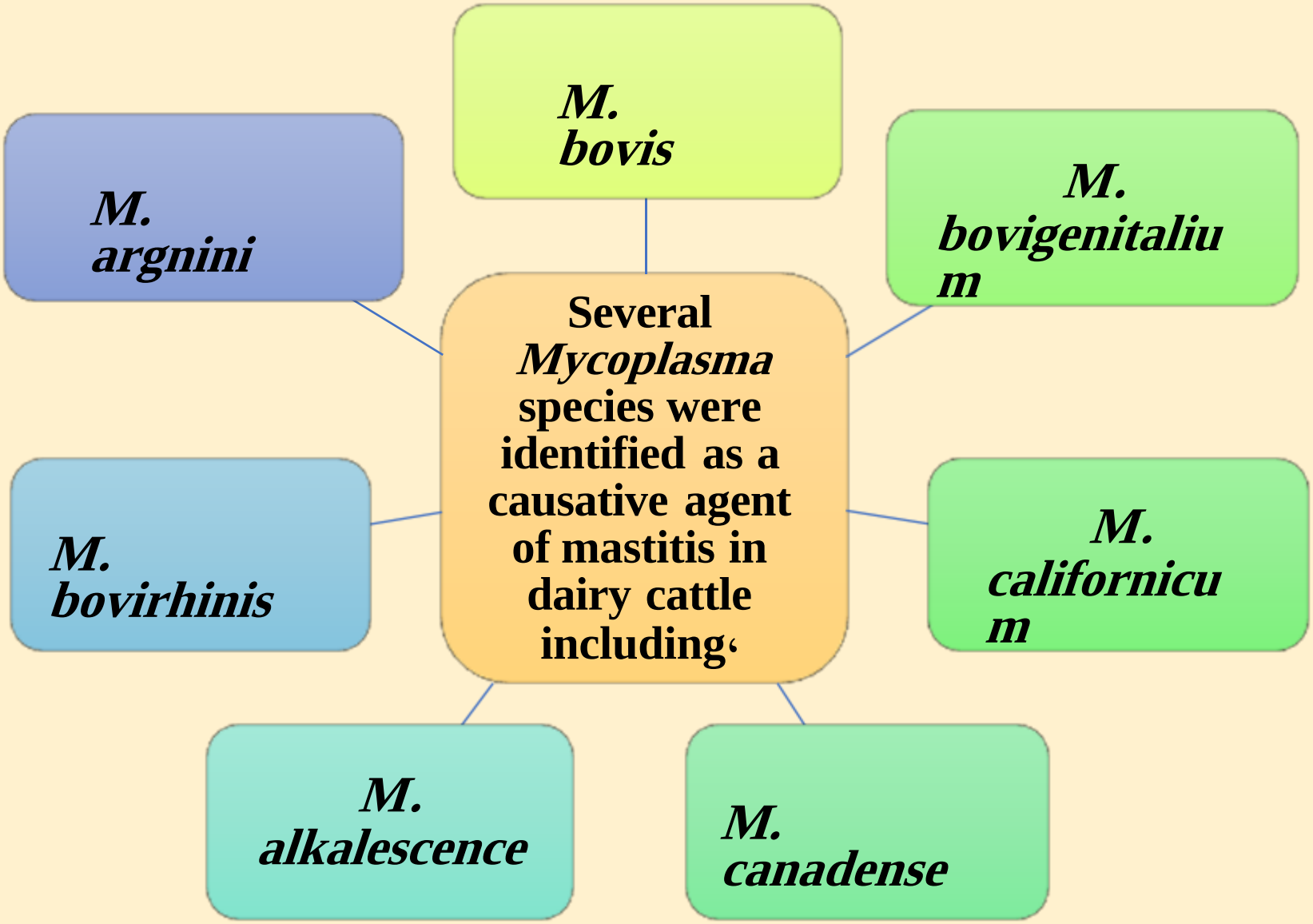
-Fluorescent antibody test.

-Indirect haemagghitination test.

-Agglutination tests.

Pathogenicity of *Mycoplasma*:

- ***M. mycoides*: causes pleuropneumonia in cattle and goats**
- ***M. agalactiae* causes contiguous agalactia (mastitis) in sheep and goats the organism are also pathogenic for dogs، rats, cats and birds.**
- **In human, *M. hominis* causes pleuropneumonia and inflammation of the urogenital tract particularly in female.**


M. bovis
M. argnini
M. bovigenitalium
Several Mycoplasma species were identified as a causative agent of mastitis in dairy cattle including‘
M. bovirhinis
M. californicum
M. alkalescence
M. canadense

Laboratory diagnosis:

-1Isolation and identification of *Mycoplasma*:

Isolation and identification of the organism by centrifugation the culture and take the sediment, which was stained with giemsa's stain and observe the typical organism.

)Morphological characters previously discussed(

-2Cultural characters and biochemical reactions:

)previously discussed(

-3Serotyping of Mycoplasma isolates.

-4ELISA

-5Molecular identification.

Immunization and control:

Experimental vaccines against Mycoplasma mastitis were ineffective; even it may worsen the condition. cows that repeatedly suffer from clinical mastitis or become negative upon culturing usually remain subclinical carriers with intermittent shedding of Mycoplasma microorganisms, and should be regarded as permanently infected.

Thank you

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

Pure microbiology

Branches of microbiology

Bacteriology: the study of bacteria.
Mycology: the study of fungi.
Protozoology: the study of protozoa.
Phycology/algology: the study of algae.
Parasitology: the study of parasites.
Immunology: the study of the immune system.
Virology: the study of viruses.
Nematology: the study of nematodes.

https://en.wikipedia.org/wiki/Branches_of_microbiology

GENERAL

BACTERIOLOGY, IMMUNOLOGY, MYCOLOGY

INTRODUCTION TO MICROBIOLOGY

MICROBIOLOGY

IS DEFINED AS

(THE STUDY OF ORGANISMS TOO SMALL TO BE CLEARLY SEEN BY THE NAKED EYES).

THESE ORGANISMS INCLUDE VIRUSES, BACTERIA, ALGEA, FUNGI, ARCHAEA AND PROTOZOA. WHEN STUDYING MICROORGANISMS , MICROBIOLOGISTS ARE CONCERNED WITH CHARACTERISTICS AND FUNCTIONS SUCH AS MORPHOLOGY ,CYTOLOGY, PHYSIOLOGY , ECOLOGY , TAXONOMY , GENETICS , AND MOLECULAR BIOLOGY .

TAXONOMY التصنيف الحيوى ECOLOGY علم البيئة

Leeuwenhoek
(Live 1632 -1723)
is the first person to use the microscopes to observe microbes: he made small handheld microscopes: he called ((microorganisms as animalcules)).

خلايا بدائيات النوى خلية بدائية النواة Procaryotes

TAXONOMY AND CLASSIFICATION OF PROCARYOTES

HEAKEL (1866) WAS THE FIREST TO CREATE ANATURAL KINGDOM FOR MICROORGANISMS , HE PLACED ALL UNICELLULAR (MICROSCOPIC) IN ANEW KINGDOM CALLED PROTISTA , SEPERATED FROM PLANTS (PLANTAE) AND ANIMALS (ANIMALIA) , WHICH WERE MULTICELLULAR (MACROSCOPIC) ORGANISMS .

PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS
PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS
ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

IN (1950) : THE DEVELOPMENT OF ELECTRON MICROSCOPE

THE ELECTRON MICROSCOPE MORE ONE MILLION POWERFUL THAN THE LIGHT MICROSCOPE.

SO BY THE ELECTRON MICROSCOPE THEY FIND ANOTHER KINGDOM CALLED MONERA (MONERES) : INCLUDES MICROSCOPIC UNICELLULAR ORGANISMS BUT NOT HAVE THE NUCLEAR MEMBRANE .

PROTISTA :
REMAINED AS AKINGDOM OF UNICELLULAR EUCARYOTIC MICROORGANISMS .

WHITTAKER : REFINED THE ECOSYSTEM INTO FIVE KINGDOMS IN (1967) BY IDENTIFYING THE FUNGI AS ASEPERATE MULTICELLULAR EUOCARYOTIC KINGDOM OF ORGANISMS , DISTINGUISHED BY THEIR ABSORPTIVE MODE OF HETEROTROPHIC MODE OF NUTRITION .

يتكون النظام البيئى إلى خمس مملكات حيوية :

THE FIVE KINGDOMS OF THE ECOSYSTEM :

1-PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS

2-PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS

3-ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

4- MONERA : UNICELLULAR MICRORGANISMS NOT HAVE NUCLEAR MEMBRANE.

5- FUNGI : MULTICELLULAR OF EUCRAYOTIC ORGANISMS ITS LENGHTH 4 MICROMETERS IN LENGTH .

BACTERIA IS 1 MICROMETERS IN LENGTH

FUNGI IS 4 MICROMETERS IN LENGTH

FIVE KINGDOMS OF THE ECOSYSTEM AS FELLOW :

ANIMALIA :INCLUDES VERTEBRATES AND ARTHROPODES

PLANTEA : INCLUDES GREEN ALGEA

FUNGI : INCLUDES MOLDS AND YEASTS

MONERA : INCLUDES ARCHAEA ,BACTERIA,MYCOPLASMA

PROTESTA : INCLUDES RED ALGEA , AMOEBAS, WATER MOLDS .

What are 5 characteristics of fungi?

Characteristics of Fungi

Fungi are eukaryotic, non-vascular, non-motile and heterotrophic organisms.

They may be unicellular or filamentous.

They reproduce by means of spores.

They Have Two Types Of Reproduction Sexually And Asexually Reproduction .

Fungi exhibit the phenomenon of alternation of generation.

Fungi lack chlorophyll and hence cannot perform photosynthesis.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

BACTERIAL MORPHOLOGY

PLEASE CHANGES THE PICTURES TO BE ELECTRONIC MICROSOPIC RECOGNITIONS

PROKARYOTIC CELLS ARE GENERALLY MUCH SMALLER THAN EUKARYOTIC CELLS

BACTERIA REMARKKABLY VARY IN SIZES , SHAPES AND ARRANGEMENT

MOST OF BACTERIA COME IN BASIC FOUR SHAPES :

1-COCCI 2- ROD OR BACILLI 3-ARCH

4-SPIRAL

1-COCCUS

THE COCCI ARE SPHERICAL OR OVAL BACTERIA HAVING ONE OF SEVERAL DISTICNT ARRANGEMENTS :

1-DIPLOCOCCUS :PAIR OF COCCI

2-STREPTOCOCCUS : CHAIN OF SEPERATE COCCI

3-TETRAD : SQUARE OF FOUR COOCI

4-SARCINA :CUBE OF 8 COCCI

5-STAPHYLOCOCCUS:COCCI IN IRREGULAR , OFTEN GRAP LIKE CLUSTE

2-BACILLUS :

BACILLI ARE ROD SHAPED BACTERIA , THEY DEVIDED IN ONE PLANE PRODUCING ONE OF THE FOLLOWING :

1-BACILLIUS : ASINGLE BACILLUS (CYLINDRICAL)

2- COCCO BACILLUS HAVE ABULGING MOSTLY IN THE MIDDLE

3-STREPTO BACILLI : ACHAIN OF SEPERATED BACILLUS TO BE FACILITATING CONGUGATION

4-SPIRAL : AHELICAL OR CORKSCREW –SHAPED BACTERIUM .SPIRALS COME IN 1 OF 3 FORMS :

لولبى

1-VIBRIO : CURVED OR COMA SHAPED ROD

2-SPIRILLUM : THICH RIGID SPIRAL

3-SPIROCHETE : THIN FLEXIBLE SPIRAL

PROCARYOTIC CELL STRUCTURES AND FUNCTIONS

THE WORD PROCARYOTE WILL BE USED IN AGENERAL SENSE TO INCLUDE BOTH BACTERIAE AND ARCHAEA
(MYCOPLASMA IN ASEPERATE TAXONOMY)
ARCHAE ARE MICROORGANISMS THEY ARE NO MORE RELATED TO BACTERIA THAN TO EUKARYOTES . THEY SHARE SOME TRAITS WITH BACTERIA ,SUCH AS HAVING ASINGLE CIRCULAR MOLECULE OF DNA ,THE PRESCENSE OF MORE MOBILE PIECES OF GENATIC MATERIALES CALLED PLASMIDS ,SIMILAR ENZYMES FOR PRODUCING COPIES OF DNA . HOWEVER THEIR METHODS OF PROTIEN PRODUCTION AND ORGANIZATION OF THEIR GENATIC MATERIAL BEARS MORE SIMILARITY TO EUBACTERIAL CELLS .
ARCHAE INCLUDES MORE ARELATIVELY SMALL NUMBERS OF MICROORGANISMS THEY ARE INHABITING ENVIRONMENTS WHICH ARE TOO HARSH FOR OTHER MICROBES.SUCH AS ENVIRONMENTS INCLUDES HOT ,MOLTEN VENTS AS AT THE BOTTOM OF OCEANS , AND THE HIGHLY SALT WATER OF THE GREAT SALT LAKE AND THE DEAD SEA.

البحر الميت – البحيرة المالحة الأمريكية العظيمة ولاية أوتاها كلاهما مالحان

فتحات مصهورة --- لقدذكرت من علامات الساعة

الكبرى جفاف بحيرة طبريا وهى من أعذب وأحلى مياه العالمين

البحر المميت الأردن قوم لوط العرب

البحيرة المالحة الأمريكية بولاية أوتاها ليس لها مسمى فاحش

UUWD89J308ULRP4

الشكوى الإدارية فى ملفات الرقابة الإدارية المصرية

DIFFERENCES BETWEEN PROKARYOTE AND EUKARYOTE AND ARCHAEA :

THE COMPARISONS WILL COVER THIS POWER POINTS :

1-CELL CONFIGURATIONS 2-NUCLAR MEMBRANE 3-NUMBER OF CHROMOSOME 4-CHROMOSOME TOPOLOGY 5-MURIEN IN CELL WALL 6-CELL MEMBRANE LIPIDS 7- CELL MEMBRANE STEROLS 8- ORGANELLES ESPECIALLY MITOCHONDRIA AND CHLOROPLASTS) 9-RIBOSOMES SIZES 10- CYTOPLASMIC STREAMING 11-MEIOSIS AND MITOSIS) 12- TRANSCRIPTIONS AND TRANSLATIONS COUPLED 13- AMINO ACID INITIATING PROTEIN SYNTHESIS

14- PROTEIN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHENICOL

15- PROTEIN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN

DIFFERENCES BETWEEN EUKARYOTE, PROKARYOTE,ARCHAEA			
PROPERITY	BIOLOGICAL DOMAIN		
	EUCARYA	BACTERIA	ARCHAEA
CELL CONFIGURATION	EUCARYOTIC	PROCARYOTIC	PROCARYOTIC
NUCLEAR MEMBRANE	PRESENT	ABSENT	ABSENT
NUMBER OF CHROMOSOMES	MORE THAN ONE	ONE	ONE
CHROMOSOME TOPOLOGY	LINEAR	CIRCULAR	CIRCULAR
MUREIN IN CELL WALL	-	+	-
CELL MEMBRANE LIPIDS	ESTER-LINKED GLYCERIDES UNBRANCHED POLYUNSATURATED	ESTER-LINKED GLYCERIDES: UNBRANCHED SATURATED OR MONOUNSATURATED	ETHER –LINKED BRANCHED: SATURATED
CELL MEMBRANE STEROLS	PRESENT	ABSENT	ABSENT

ORGANELLES (MITOCHONDERIA AND CHLOROPLASTS)	PRESENT	ABSENT	ABSENT
RIBOSOME SIZE	80S	70S	70S
CYTOPLASMIC STREAMING	+	–	–
MEIOSIS AND MITOSIS	PRESENT	ABSENT	ABSENT
TRANSCRIPITION AND TRANSLATION COUPLED	–	+	+
AMINOACID INITIATING PROTIEN SYNTHESIS	METHIONINE	N-FORMYLE METHIONINE	METHIONINE
PROTIEN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHINCOL	–	+	–
PROTIEN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN	+	–	+

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

BACTERIAL STRUCTURES

INCLUDES :

1- ESSENTIAL BACTERIAL STRUCTURES

2- NON ESSENTIAL BACTERIAL STRUCTURS

1- ESSENTIAL BACTERIAL STRUCTURES

INCLUDES :

1-CELL WALL AND

2-PROTOPLASM

2-PROTOPLASM INCLUDES :

A-CYTOPLASM AND

B-CELL MAMBRANE

A-CYTOPLASM INCLUDES :

CHROMOSOMES ,MESOSOMES,RIBOSOMES, INCLUSIONS BODIES.

2-NON ESSENTIAL BACTERIAL STRUCTURES :

INCLUDES :

CAPSULES,FLAGELLAE,PILI,
SPORES,
PLASMIDES.

1-ESSENTIAL BACTERIAL STRUCTURES :

1-CELL WALL :

1-ONE OF THE MOST IMPORTANT PARTS OF THE PROKAYOTIC CELL.

2-MOST OF PROCAYOTES HAVE ARIGID CELL WALL.

3-THE CELL WALL IS CELL MADE OF PEPTIDOGLYCANS: POLYMER OF PEPTIDES AND GLYCANS.

PEPTIDES (TYPICAL 4 AMINOACIDES LONG ,CROSS-LINKED TO OTHER CHAINS)

GLYCANS (MADE OF ALTERNATING AMINO SUGARS)

4- PEPTIDOGLYCAN IS AHUGE POLYMER OF INTERLOCKING CHAINS OF IDENTICAL MONOMERS.

5- THE BACKBONE OF PEPTIDOGLYCAN MOLECULES COMPOSED OF TWO DERIVATIVES OF GLUCOSE :
N-ACETYLE GLUCOSAMINE(NAG)
AND N-ACETYLMURAMIC ACID (NAM)
THE (NAG) AND (NAM) STRANDS ARE CONNECTED BY INTERPEPTIDE BRIDGES

6-FUNCTIONS OF CELL WALL :

1-SUPPORTS THE WEAK CYTOPLASMIC MEMBRANE AGAINST INTERNAL OSMOTIC PRESSURE .

2-RESPONSIBLE FOR THE SHAPE OF BACTERIAE AND THEIR GRAM STAIN REACTIONS .

3-INTEGRITY OF THE CELL WALL ESSENTIAL FOR VIABILITY OF BACTERIAE .

4-PLAY ROLE IN CELLS DIVISIONS AND ANTIGENICITY OF THE BACTERIAE .

IN THE GRAM POSITIVE BACTERIAE THOSE RETAIN CRYSTAL VIOLET DYE WHEN SUBJECTED TO THE GRAM STAINING PROCEDURES , THE CELL WALL CONSISTS OF SEVERAL LAYERS OF PEPTIDO GLYCAN . TEICHOIC ACIDS RUNING PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.
TEICHOIC ACIDS IS AGROUP OF MOLECULES WHICH ARE UNIQUE TO GRAM POSITIVE CELL WALL .

STRUCTURE OF THE GRAM POSITIVE BACTERIAL CELL WALL .THE CELL WALL IS RELATIVELY THICK AND CONSISTS OF MANY LAYERS OF PEPTIDO GLYCANS INTERSPERSED WITH TECHOIC ACIDS THAT RUN PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.

IN THE GRAM NEGATIVE BACTERIAE (WHICH DO NOT RETAIN CRYSTAL VIOLET DYE EXCEPT ALITTLE) :

THE CELL WALL COMPOSED OF ASINGLE LAYER OF PEPTIDO GLYCANS SURROUNDED BY AMEMBRANOUS STRUCTURE CALLED THE OUTERMEMBRANE.

THE OUTER MEMBRANE OF GRAM NEGATIVE BACTERIAE INVARIABLY CONTAINS AUNIQUE COMPONENT,LIPOPOLYSACCHARIDES (LPS OR ENDOTOXIN), WHICH IS TOXIC IN ANIMALS.

STRUCTURE OF THE GRAM NEGATIVE- BACTERIAE CELL WALL.

THE WALL IS RELATIVELY THIN AND CONTAINS MUCH LESS PEPTIDO GLYCANS THAN THE GRAM POSITIVE BACTERIAL WALL. ALSO,TEICHOIC ACIDS ARE ABSENT. HOWEVER, THE GRAM NEGATIVE-CELL WALL CONSISTS OF AN OUTERMEMBRANE THAT IS OUTSIDES OF THE PEPTIDO GLYCAN LAYER.THE OUTERMEMBRANE IS ATTACHED TO THE PEPTIDO GLYCAN SHEET BY AUNIQUE GROUP OF LIPOPROTIEN MOLECULES.

THE PROFILES OF THE CELL ENVELOPE OF THE GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE.

THE GRAM POSITIVE WALL IS AUNIFORMLY THICK LAYER EXTERNAL TO THE PLASMA MEMBRANE . IT IS COMPOSED MAINLY OF PEPTIDOGLYCAN (MEURIN).

THE GRAM NEGATIVE WALL

APPEARS THIN AND MULTILAYERED.

IT IS CONSIST OF ARELATIVELY THIN PEPTIDOGLYCAN SHEET BETWEEN THE PLASMA MEMBRANE AND APHOSPHOLIPID-LIPOPOLYSACCHARIDE OUTERMEMBRANE.

THE SPACE BETWEEN THE INNER PLASMA MEMBRANE AND OUTER MEMBRANE WHERE IN THE PEPTIDOGLCAN RESIDES IS CALLED PERIPLASM.

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

1-BACTERIAL MEMBRANES COMPOSED OF 40% TO 60% PROTIEN.BACTERIAL PLASMA MEMEBRANES USUALY HAVE AHIGHER PROPORTION OF PROTIEN THAN DO EUCARYOTIC MEMBRANES,PERSUMABLY BECOUSE THEY FULFIL SO MANY DIFFERENT FUNCTIONS THAT ARE CARRIED OUT BY OTHER ORGANELLE MEMBRANES IN EUCARYOTES.

FUNCTIONS OF-Be- CELL MEMBRANE:

1-OSMOTIC BARRIER FOR MAINTAINING DIFFERENCE IN SOLUTE CONTENT BETWEEN CYTOPLASM AND EXTERNAL ENVIRONMENT.

2-PERMIT PASSIVE DIFFUSION INWARD FROM OUTWARD OF WATER AND LIPID SOULABLE MATERIAL.

3-SELECTIVE TRANSPORT OF SPECIFIC NUTRIENT SOLUTE IN TO THE CELL.

4-ENZYMATIC ACITIVITY AS IT CONTAINS PERMEASE,RESPIRATORY,CYTOCHROME AND POLYMERZING ENZYMES.

5-ENERGY GENERATING FUNCTIONS,INVOLVING RESPIRATORY AND PHOTOSYNTHETIC ELECTRON TRANSPORT SYSTEMS,ESTABLISHMENT OF PROTON MOTIVE FORCE, AND TRANSMEMBRANOUS, ATP-SYNTHESIZING ATPase.

6-COORDINATIONS OF DNA REPLICATIONS AND SEGREGATION WITH SEPTUM FORMATION AND CELL DIVISION.

2-THE PERIPLASM :

IS ASPACE BETWEEN PLASMA MEMBRANE AND CELL WALL.

IT IS VISIBLE IN GRAM NEGATIVE AND DIFFICULT IN GRAM POSITIVE BACTERIAE.

IT CONTAIN VARIOUS HYDROLYTIC ENZYMES AND BINDING PROTIENS THAT SPECIFICALLY BIND SUGARS ,AMINOACIDS AND INORGANIC IONS ,THESE IONS AND PROTIENS THAT AID IN TRANSPORT VARIOUS COMPOUNDS INTO AND OUT BACTERIAL CYTOPLASM.

3-THE CYTOPLASM :

1-THE CYTOPLASM OF BACTERIAL CELL IS VISCOUS WATERY SOLUTION CONTAIN ORGANIC AND INORGANIC SUBSTANCES AND RIBOSOMES.
2-IT HAS NO MITOCHONDERIA,NO ENDOPLASMIC RETICULUM, NO GOLGI APPARATUS.
3-IT COMPOSED FROM RNA.
4-IT CONTAINS BACTERIAL CHROMOSOMES (NECLEOID),RIBOSOMES,INCLUSION GRANULES AND MESOSOMES.

4-THE BACTERIAL CHROMOSOMES (NECLOID):

IT COMPRISES ACLOSED LOOP OF DOUBLE STRANDED SUPERCOILED DNA.(NO NUCLEAR MEMBRANE).IN ADDITION ,SOME BACTERIAE CONTAIN ADDITIONAL DNA IN THE FORM OF SMALL ,SELF REPLICATING EXTERA-CHROMOSOMAL ELEMINTS CALLED PLASMIDS.THESE DO NOT CARRY ANY GENES ESSENTIAL FOR GROWTH AND REPRODUCTIONS,AND THUS THE CELL MAY SERVIVE WITHOUT THEM. THE PLASMIDS CAN BE VERY IMPORTANT HOWEVER,AS THEY MAY INCLUDE GENES ENCODING TOXINS OR RESISTANCES TO ANTIBIOTICS ,AND CAN BE PASSED FROM CELL TO CELL.

5-RIBOSOMES:

THESE ARE THE SITES OF PROTIENS SYNTHESIS.ALTHOUGH THEY CARRY OUT ASIMILAR FUNCTIONS,THE RIBOSOMES OF PROCARYOTIC CELLS ARE SMALLER AND LIGHTER THAN THIER EUCARYOTIC CELL.
PROCARYOTIC RIBOSOMES ARE 70 S ,WHILE THOSE OF EUCARYOTES ARE 80 S.

6-MESOSOMES:

IT IS ACONVOLUTED BODY INVAGINATED FROM CYTOPLASMIC MEMBRANE INTO CYTOPLASM.
1-ENTER IN THE MECHANISM OF CELL DIVISION.
2-HAS AROLE IN SPORULATION OF BACTERIA.
3- ACT AS AMITOCHONDERIA OF EUCARYOTIC CELL.
4-HAS AROLE IN RESPIRATION OF BACTERIA.
5-EXCRETE MATERIALS FROM CYTOPLASM TO EXTERIOR.

7-INCLUSIONS BODIES :

1-IT WERE ASTORED RESERVES OF NUTRIENTS OF AGGREGATES OF EXCESS SUBSTANCES .

2-IT VARRIES ACCORDINDG TO TYPE OF BACTERIAL STRUCTURE OR GENOMES.

E.G: FOR EXAMPLES:

a-VOLUTIN (METACHROMIC GRANULES) IN PASTEURELLA SPP.

b-LIPIDS GRANULES IN TUBERCLE BACILLI.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

NON ESSENTIAL STRUCTURES

1-CAPSULES AND -S LAYERS

2-FELAGELLAE

3-PILI

4-SPORE

5-PLASIMDS .

1-CAPSULES AND S LAYERS :

A-SOME BACTERIA HAVE ALAYER OF MATERIAL LYING OUTSIDE THE CELL WALL

B-WHEN THE LAYER IS WELL RECOGNIZED AND EASILY WASHED OFF IT IS CALLED ATRUE CAPSULE.

C-ATRUE CAPSULE IS A DISCRETE DETECTABLE LAYER OF POLYSACCHARIDES DEPOSITED OUTSIDE THE CELL WALL.

D-ALESS DISCRETE STRUCTURE OR MATRIX WHICH EMBEDS THE CELL CALLED ASLIME LAYER OR BIOFILM.

E-CAPSULES AND SLIME LAYERS USUALLY ARE COMPOSED OF POLYSACCHARIDES .

F-BUT THEY MAY BE CONSTRUCTED FROM OTHER MATERIALS .

G-FOR EXAMPLE BACILLUS ANTHRAX HAVE ACAPSULE OF POLY-GLUTAMIC ACIDS .

H-CAPSULES ARE CLEARLY VISIBLE BY IN LIGHT MICROSCOPE.

I-MANY GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE HAVE AREGULARLY STRUCTURED LAYER AN S LAYER ON THIER SURFACE . THE S-LAYER HAS APATTERN SOMETHING LIKE FLOOR TILES AND IS COMPOSED OF PROTIEN OR GLYCOPROTIEN.

N.B THERE AN ACONFESSION THAT THERE AN ELECTRON MICROSOPE IMPLENTATIONS IN THE UNIVERSITY BOOK.

J-BACTERIAL CAPSULES OUTLINED BY INDIA INK VIEWED BY LIGHT MICROSCOPY .THIS ATRUE CAPSULE ,ADISCRETE LAYER OF POLYSACCHARIDE SURROUNDING THE CELLS.SOMETIMES BACTERIAL CELLS ARE EMBEDDED MORE RANDOMLY IN APOLYSACCHARIDES MATRIX IN ASLIME LAYER OR BIOFILM.

FUNCTIONS OF CAPSULES :

A-PROTECT THE CELL WALL AGAINST ANTIBACTERIAL AGENTS .
B-PROTECT THE BACTERIAL CELLS AGAINST INGESTION BY THE PHAGOCYTIC CELLS .
C-RESPONSIBLE OF VIRULENCE OF THE ORGANISMS
D-THE S -LAYER CAN FUNCTION AS AN ADHESION ,ENABLING THE BACTERIUM TO ADHERE TO HOST CELL MEMEBRANES AND ENVIRONMENTAL SURFACES IN ORDER TO COLONIZES .
E-MANY OF THE CELL –ASSOCIATED PROTIEN ADHESINS USED BY PATHOGENS ARE COMPONENTS OF THE S-LAYER.

2-FLAGELLA :

A-MOST MOTILE BACTERIA MOVE BY USE OF FELAGELLA (S.FLALGELLUM) ,
B-THREADS LIKE LOCOMOTOR APPENDAGES EXTENDING OUTWARD FROM THE PLASMAMEMBRANE.
C-THEY ARE SLENDER , RIGID STRUCTURES ,ABOUT 20 NM ACROSS AND UP TO 15 OR 20 MICRO METER LONG .
D-FLAGELLA ARE SO THIN THEY CAN NOT BE OBSERVED DIRECTLY WITH –A BRIGHT FIELD MICROSCOPE.
E-BUT MUST BE STAINED WITH ESPECIAL TECHNIQUES DESIGNED TO INCREASE THEIR THICKNESS.

F-BACTERIAL SPECIES OFTEN DIFFER DISTINCTIVELY IN THEIR PATTERNS OF FLAGELLAE DISTRIBUTIONS :

1-MONOTRICHOUS BACTERIA (TRICHOUS MEANS HAIR) HAVE ONE FELAGELLUM:IF IT IS LOCATED AT THE END IT IS SAID TO BE POLAR FLAGELLUM

2-AMPHITRICHOUS BACTERIA (AMPHI MEANS ON BOTH SIDES) LE FELAGELLUM ز HAVE ASINGLE FELAGELLUM ON THE BOTH POLE. ON THE BOTH POLE>BOTH POLE BE POLAR FLAGELLUM E PLASMAMEMBRANE MEBRANE LE ,ADISCRETE LAYER OF POLYSACCHARIDE SURR

3-LOPHOTRICHOUS BACTERIA (LOPHO MEANS TUFT) HAVE ACLUSTER OF FELAGELLA AT ONE OR BOTH ENDS .

4-PERITRICHOUS (PERI MEANS AROUND) BACTERIA HAVE FLAGELLA THAT ARE SPREAD FAIRLY EVENLY OVER THE WHOLE SURFACE . FELLAGELATION PATTERNS ARE VERY USEFUL IN IDENTIFYING BACTERIA.

G-FELAGELLAR ULTRASTRUCTURE:

A-THE LONGEST AND MOST OBIVIOUS PORTION IS THE FILAMENT ,WHICH EXTENDEDS FROM THE CELL SURFACE TO THE TIP.

b-abasal body is embedded in the cell :the body has four rings connected to a centeral rod . the outer l and p rings associated with the lipopolysaccharides and peptidoglycan layers ,respectively .

d-ashort curved segment ,the hook ,links the filament to to its basal bodies and acts as aflexible coupling.

THE FILAMENT IS AHOLLOW ,RIGID CYLINDER CONSTERUCTED OF ASINGLE PROTIEN CALLED FLAGELLIN .

3-FIMBRIAE AND PILI :

FIMBRIAE AND PILLI ARE SHORT-HAIR LIKE STRUCTURES ON THE SURFACES OF PROCARYOTIC CELLS .LIKES FELAGELLA ,THEY ARE COMPOSED OF PROTIEN .FIMBRIAE ARE SHORTER AND STIFFER THAN FELAGELLA AND SLIGHTELY SMALLER IN DIAMETER .GENERALLY ,FIMBERIAE HAVE NOTHING TO DO WITH BACTERIAL MOVEMENT (THERE ARE EXCEPTIONS, E.G . TWITCHING, MOVEMENT ON PSEUDOMONAS).

N.B FIMBERIAE ARE MOST COMMON IN GRAM NEGATIVE BACTERIAE , BUT OCCUR IN SOME GRAM POSITIVE BACTERIAE AS WELL.

FIMBRIAE ARE MOST OFTEN INVOLVED IN ADHERENCE OF BACTERIA TO SURFACES ,SUBSTRATES AND OTHER CELLS OR ISSUES IN NATURE. IN E.CLOI , ASPECIALIZED TYPE OF PILUS , THE F OR SEX PILUS, APPARENTELY STABLIZES MATING BACTERIA DURING THE PROCESS OF CONJUGATION , BUT THE FUNTION OF THE SMALLER , MORE NUMEROUS COMMON PILLI ARE QUITE DIFFERENT .

COMMON PILI (ALMOST ALWAYS CALLED FIMBRIAE) ARE USUALLY INVOLVED IN SPECIFIC AHERENCE (ATTATCHMENT) OF PROCARYOTES TO SURFACES IN NATURE.

IN THE MEDICAL SITUATIONS , THEY ARE MAJOR DETERMINANTS OF BACTERIAL VIRULENCE BECOUSE THEY ALLOW PATHOGENS TO ATTACH TO (COLONIZE) TISSUES AND OR TO RESIST ATTACK BY PHAGOCYTIC WHITE BLOOD CELLS . FOR EXAMPLE , PATHOGENIC NEISSERIA GONORHOEAE ADHERES SPECIFICALLY TO THE HUMAN CERVICAL OR URETHERAL EPITHELIUM BY MEANS OF FIMBERIAE : ENTEROTOXIGENIC STRAINS OF ESCHERICHIA COLI ADHERE TO THE MUCOSAL EPITHELIUM OF THE INTESTINE BY MEANS OF SPECIFIC FIMBRIAE .

PROPERTY	FLAGELLA	PILI
COMPOSITION	FLAGELLIN, APROTIEN	PILIN, APROTIEN
OCCUERENCE	MANY BACTERIAL RODS,BUT FEW COCCI	MANY GRAM NEGATICE AND COCCI
FUNCTION	MOTION	CONJUGATION,ATTACHMENT
SHAPE	LONG AND THIN	SHORT AND STIFF
ORIGIN	HOOK LIKE INSERTION TO BASAL GRANULE INSIDE CELL WALL	CYTOPLASM

4- THE BACTERIAL END SPORES:

1-ANUMBER OF GRAM POSITIVE BACTERIA CAN FORM ASPECIAL RESISTANT , DORMANT STRUCTURE CALLED AN END SPORES .
2-END SPORES DEVELOP WITHIN VEGETATIVE BACTERIAL CELLS OF SEVERAL GENERA
3-SEVERAL GENERA INCLUDES : 1- BACILLUS LIKES ANTHRAX AND CLOSTRIDIUM (RODS) AND 2- COCCI LIKES SPOROSARCINA
3- AND OTHERS .

4- THESE STRUCTURES ARE EXTRA ORDINARILY RESISTANT TO INVIRONMENTAL STRESSES SUCH AS HEAT,ULTRA VIOLET, RADIATION,GAMMA RADIATIONS, CHEMICAL DISINFECTANTS,DESICCATIONS,

5- IN FACTS ,SOME ENDO SPORES HAVE REMAINED VIABLES FOR AROUNDING 100,000 YEARS.

6-BECOUSES OF THEIR RESISTANSE AND INFACT THAT SEVERAL SPECIES OF ENDOSPORES –FORMING BACTERIA ARE DANGEROUS PATHOGENS , ENDOSOPRES ARE OF GREAT PRACTICAL IMPORTANCE IN FOOD,INDUSTERIAL ,AND MEDICAL MICROBILOGY ,

7-THIS BECOUSE ENDOSPORES OFTEN RESIST SURVIVE BOILING FOR AN HOUR OR MORE.

8-SPORES POSITIONS IN THE MOTHER CELLS , (SPORANGIUM) FREQUENTLY DIFFERS AMONG SPECIES ,MAKING IT OF CONSIDERABLE VALUE IN INDENTIFICATIONS , AS IT MAY LOCATE CENTRAL, TERMINAL OR SUBTERMINAL .

9- SPORE STRUCTURES :

A-THE SPORES OFTEN IS SURROUNDED BY ATHIN DELICATE COVERING CALLED THE EXOSPORIUM .

B-ASPORE COAT LIES BENEATH THE EXOSPORIUM ,IS COMPOSED OF SEVERAL PROTIENS LAYERS AND MAY BE FAIRLY THICK, IT IS IMPERMEABLE AND RESPONSIBLE FOR SPORES RESISTANCES TO CHEMICALS .

C-THE CORTEX, WHICH MAY OCCUPY AS MUCH AS HALF THE SPORE VOLUME , RESISTS BENEATH THE SPORE COAT IT WERE MADE OF PEPTIDO GLYCANS .

D-THE SPORE CELL WALL (OR CORE WALL) IS INSIDES THE CORTEX AND SERROUNDS THE PROTOPLASTS OR CORES .

E- THE CORES HAS THE NORMAL CELL STRUCTURES SUCH AS RIBOSOMES AND ANEUCLOIDES , BUT IT METABOLICALLY INACTIVE.

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :بكر

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :

1- 15 % OF THE SPORES DRY WEIGHT CONSISTS OF DIPICOLINC ACIDS COMPLEXED WITH CALICIUM IONS WHICH IS LOCATED IN THE CORES. IT HAS LONG BEEN THOUGHT THAT DIPICOLINIC ACIDS WAS DIRECTLY INVOLVED IN SPORES HEAT RESISTANCES .

2- DEHYDERATIONS OF THE PROTOPLASTS VERY IMPORTANT IN HEAT RESISTANCES .

3- THE CORTEX MAY OSMOTICALLY REMOVE WATER FROM PROTOPLASTS ,THERBY PROTECTING IT FROM HEAT AND RADIATIONS DAMAGES.

4-THE SPORES COATS ALSO SEEMS TO PROTECTING AGAINST ENZYMES AND CHEMICALS SUCH AS HYDROGEN PEROXIDES .

11- SPORE FORMATIONS (SPOROGENESIS OR SPORULATIONS):

1-NORMALLY COMMENCES WHEN GROWTH CEASES DUE TO LACK OF NUTRIENTS . IT IS ACOMPLEX PROCESS AND MAY DEVIDED INTO SEVEN STAGES :

1-AN AXIAL FILAMENT OF NUCLEAR MATERIAL FORMS .

2-INWARD FOLDING OF THE CELL MEMBRANE TO ENCLOSE PART OF THE DNA ORODUCE THE FORESPORE SEPTUM .

3-THE MEMBRANE CONTINUES TO GROW TO ENGULFS THE IMMATURE SPORE IN THE SECOND MEMBRANE.

4-CORTEX IS LAID DOWN IN THE SPACE BETWEEN THE TWO MEMBRANES ,AND BOTH CALCIUM AND DIPICOLINIC ACIDS ARE ACCUMULATED .

5-PROTIEN COATS THEN ARE FORMED AROUND THE CORTEX .

6-MATURATIONS OF THE SPORE OCCURS.

7-LYTING ENZYMES DESTROY THE SPORANGIUM RELEASING THE SPORE.

SPORULATION REQIURES ONLY ABOUT TEN HOURS IN BACILLUS MEGATERIUM .

JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS

E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :

1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM

IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
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الباقل الكلوى المزمن

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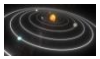
الوصول إلى الشرف للإحاطة.pdf



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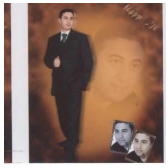

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الجمعية الوطنية للقلب والباطنة الكلوية وأمراضه الصحية

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

التصنيفات للبكتيريا المهمة طبيا

جامعة بنها

كلية الطب البيطرى

قسم البكتيريا والمناعة والفطريات

كتبهاونسقها مساعدباحث محمدمحمودمحمديوسف وهيبه

ماجيستير البكتيريا والمناعة والفطريات

Classification of medically important bacteria

1- RIGID, THICK- WALLED CELLS	2- FLEXIBL, THIN WALLED CELLS	3- WALL LESS – CELLS
	Leptospira	**Mycoplasma**

1- RIGID, THICK- WALLED CELLS

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)	B- NON-FREE-LIVING BACTERIA (OBLIGATE INTERACELLAR BACTERIA (as Rickettsia and Chlamydia)

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)		
1- Gram positive bacteria (stained appear blue colour)	2- Gram negative bacteria (not stained just Avery little appear red or pink colour)	3-Acid fast bacteria e.g T.B Mycobacterium

<table>
<tr><td colspan="3">1- Gram positive bacteria (stained appear blue colour)</td><td colspan="2">2- Gram negative bacteria (not stained just Avery little appear red or pink colour)</td></tr>
<tr><td>a- Cocci
Staph
Strept</td><td colspan="2">b- Bacilli include : Spore forming and Non Spore Forming</td><td rowspan="4">a- Cocci like Nisseria</td><td rowspan="4">b- Bacilli include : Aerobic (Psudomonas) and Facultative Anaerobic (Straight – Curved) and Anaerobic bacteria (Bacteroides)</td></tr>
<tr><td rowspan="3"></td><td>Spore forming</td><td>Non Spore forming</td></tr>
<tr><td>Aerobic bacteria like Bacillus Anthracis
Anaerobic bacteria like Clostridia</td><td>non Filamentous like
Coryn bacteria,
Listeria and Filamentous Actinomyces</td></tr>
<tr><td></td><td></td></tr>
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Facultative anaerobic

Straight			Curved
Respiratory bacteria Haemophilus	Zoonotic bacteria- Brucella , Pasteruella , Yersenia	Enteric bacteria E.coli , Salmonella, Shigella ,Klebsiella, Proteus	Campylobacter , Vibriocholera ,Hilicobacter

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

Some of Important bacterial diseases

Written and presented by

Doctor

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Some of Important bacterial diseases

Streptococcus	pneumonia , pharyngitis , cellulitis
Staphylococcus	abscess of skin and other organs
Bacillus anthraces	anthrax
Clostridium	tetanus , gas gangrene , botulism

Coryne bacterium	diphtheria
Listeria	meningitis
Actinomyces	actinomycosis

Nocardia	nocardiosis
Neisseria	gonorrhea , meningitis
Haemophilus	meningitis
Bortedella	whooping cough
Legionella	pneumonia
Brucella	brucellosis
Francisella	tularemia

Pasteurella	cellulitis
Yersinia	plaque
Escherichia coli	urinary tract infection , diarrhea
Enterobacter	urinary tract infection
Serratia	pneumonia
Klebsiella	urinary tract infection
Salmonella	enterocolitis , typhoid fever

Shigella	**enterocolitis**
Proteus	**urinary tract infection**
Campylobacter	**enterocolitis**
Helicobacter	**gastritis , peptic ulcer**
Vibrio	**cholera**
Pseudomonas	**urinary tract infection , pneumonia**
Bacteroids	**peritonitis**

Mycobacterium tuberculosis , leprosy
Rickettssia (rocky mountain spotted fever , typhus , Q fever)
Chlamydia urethritis , trachoma , psittacosis
Treponema syphilis
Borrelia lyme disease
Leptospira leptospirosis
Mycoplasma pneumonia

BENHA UNIVERSITY

FACULTY OF VETERINARY MEDICINE

DEPARTMENT OF ZOONOSES

ORGANIZED AND PRESENTED BY

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(2012)

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MINISTRY OF AGRICULTURE AND LAND RECLAMATION

ARAB REPUBLIC OF EGYPT

مجموعة ١٣١٣

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معهد بحوث صحة الحيوان بطنطا
ذكر
مسلم
أعزب
البطاقة سارية حتى ٢٠٢٩/٠٥/٢١

جمهورية مصر العربية
بطاقة تحقيق الشخصية
محمد
محمود محمد يوسف وهيبه
ش العوض -كفر الزيات
مركز كفر الزيات - الغربية
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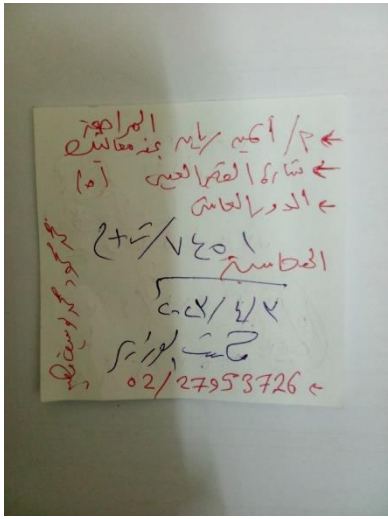
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EPIDEMYOLOGY OF BRUCELLA

DEMANDS OF LOBNA SALEM

DEPARTMENT HEAD

ZOONOSIS

BACTERIOSIS

BRUCELLOSIS

BRUCELLOSIS

1-BRUCELLOSIS IS AZOONOSIS

AWORLD WIDE DISTRIBUTION

2-ACUTE AND OR CHRONIC DISEASE

3-MAY CAUSE APHYSICAL IN CAPACITY

4- HAVE MANY SYNONYMS

4- HAVE MANY SYNONYMS INCLUDES :

A-SYNONYMS OF INFECTIONS IN HUMANITY :

-1-UNDULENT FEVER 11- MALTA FEVER 111- MEDITERRANEAN FEVER

B-SYNONYMS OF INFECTIONS IN ANIMALS :

1- CONTAGIUOS ABORTIONS IN ANIMALS

11-BANG S ABORTIONS IN CATTLE

5-CAUSTIVE ORGNISMS :

THREE MAJOR SPECIES OF BRUCELLAE INCLUDES :

1-BRUCELLA ABORTUS

2- BRUCELLA MELITENSIS

3-BRUCELLA SUIS

4-RECENTLY BRUCELLA CANIS

BRUCELLA ABORTUS OF CATTLE: CUSING ABORTION OF CATTLE AT 6-10 MONTH OF PREGENANCY

BRUCELLA MELITENSIS OF GOATS AND SHEEP : CAUSING ABORTIONS IN THESE ANIMALS AT 3 TO 4 MONTHS OF PREGENANCY BUT MORE SEVER INFECTIONS IN HUMANS AND MAY BE FATAL ESPECIALLY IN YOUNG AND ELDERLY AGES BUT ABOTION IS LESS COMMON ON ALARGE SCALES IN ANIMALS AS ACOMPARISONS OF RUMINANTS

BRUCELLA SUIS OF PIGS : CAUSING ABORTION OF DOGS ABOUT 50 DAYS OF GESTATION

BRUCELLA CANIS : GIVES COMPLETE HOMOLOGY WITH BRUCELLA SUIS

BRUCELLA CANIS RARELY INFECT MAN BUT SLIGHTLY INVASIVE FOR HIM

5-BRUCELLAE ARE RELATIVELY RESISTANCE IN THE ENVIRONMENT
6-BUT READILY DESTORYED BY PASTEURIZATIONS AND BOILING OF MILK AND CHLORINATION OF WATER
7-PATHOGENICITY :
N.B-THE 4 BRUCELLA SPECIES ARE PATHOGENIC TO MAN BUT THEIR VIRULENCE VARIES IN THE ORDER OF MELITENSIS ,SUIS , ABORTUS , CANIS.
8-RESERVOIRS OF INFECTIONS :
1-BRUCELLA IS AZOONOSES PAR EXELLENCE WHY :-
A-THE ROLES OF ANIMALS ARE ESSENTIALS IN TH E PERSISTANCES OF INFECTIONS .
B-HUMAN TO HUMAN TRANSMISSION ARE EXCPTIONAL .
C-THE ORIGINAL RESERVOIRS INCLUDE :
A-GOATS B- SHEEP C-PIGS D- DOGS
D- THE MINOR RESERVOIRS INCLUDES :
A-HORSES B- FOWEL (ARE YOU SURE) C- SOME SPECIES OF RODENTS
MAJOR AND MINOR RESERVOIRES ARE CONSIDERED AS ASOURCES OF HUMAN INFECTIONS

9- ORIGINAL RESERVIORS CAN BE INFECTED WITH OTHER SPECIES OF BRUCELLAE BESIDE THE SPECIFIC SPECIES :

E.G A- CATTLE CAN ALSO BE INFECTED BY BRUCELLA SUIS AND BRUCELLA MELITENSIS

B-GOATS AND SHEEPS CAN BE INFECTED BY BRUCELLA SUIS AND BRUCELLA ABORTUS

C-SWINE CAN BE INFECTED BY BRUCELL ABORTUS

D-DOGS CAN BE INFECTED BY BRUCELLA ABORTUS AND BRUCELLA SUIS AND BRUCELLA MELITENSIS

10-BRUCELLOSIS IN HORSES USUALLY MANIFESTS ITSELF IN THE FORM OF :

1- FISTULOUS BURSITIS (POLL EVIL) AND FISTULOUS WITHERS .

2-ABORTION ARE IN RARE IN HORSES .

3-MAN CAN CONTRACT THE INFECTIONS FROM HORSES WITH OPEN LESIONS .

11-EPIDEMYLOGICAL STUDIES OF BRUCELLOSIS DURING THE LAST DECADES PROVED THAT NOT ONLY DOMESTIC ANIMALS ARE THE MAIN CARRIERS BUT ALSO 24 SPECIES OF WILD ANIMALS AND 18 SPECIES OF BLOOD SUCKING ARTHROPODES CAN HARBOUR THE BRUCELLAE AND CATS ARE RESISTANCE TO BRUCELLA AN NO CASES OF NATURAL DISEASE OCCUERENCE ARE KNOWN

12- ORGANISMS ARE FOUND IN : EXCRETA ,DISCHARGES,TISSUES

TISSUES INCLUDING : MUSCLES ,MAMMARY GLANDS , FOETUS ,PLACENTA AND VAGINAL DISCHARGES OF ABORTED ANIMALS

13- SOURCE OF INFECTIONS AND MODE OF TRANSMISSION:

MAN IS INFECTED BY ANIMALS THROUGH :

1-DIRECT CONTACT

2-INDIRECT CONTACT INCLUDES (FOOD BORNE INFECTION AND INHALATION OF AIR BORNE AGENTS)

1-FOOD BORNE INFECTION INCLUDE :

A-INGESTION OF RAW OR INSUFFICIENTLY HEATED MILK AND MILK PRODUCTS MADE OF IT ESPECIALLY FRESH CHEESE ,ICE CREAM , CREAM AND YOUGHERT NOT COMPLETELY FERMENTED B-THE ORGANISMS RARELY SURVIVE IN SOUR MILK ,SOUR CREAM ,BUTTER AND FERMENTED CHEESES AGED BY OVER THREE MONTHS .

b-the relatively scarcity of cases among children inspite of large consumption of raw milk suggestes that certain antarceologic factors of resistance are present as (lymphoid tissues more active in childerns and gastric acidity)

c-but in adults brucella was not correlated with achlorohydria , it is presumed that the organisms pass through the mucous membranes of oropharaynx,not through the stomach (are you sure) .

d-ingestion of insufficiently cooked or processed meat

e-brucellae are resistance to pickling and smoke curing thus giving rise to the possibility that some meat products so inefficient prepared could cause human infections how ever this mode of transmission has never been verified

d-it is also possible for raw vegetables and water contaminated with the excreta of infected animals to serve as sources of infections.

2-contact infection :

a-the organisms find the entry through the skin and the mucous membranes with abrasions or intake

b-brucellae produce proteolytic enzymes which ease infections through intaked skin and mucous membranes

c-brucellosis is ,for the most part ,an occupational disease of stock yard and slaghter house workers ,,butchers , veterinarians

d-the infection is usually contracted by handling fetuses and after birth ,or by contact with vaginal secretions ,excreta, and carcasses of infected animals .

e-contact infections from horses with open lesions of fistulous bursitis must be not ignored

f-contact infections in laboratories is possibles

g-several veterinarians accidentally inoculated during vaccinations of heifers with strain -19 of brucella abortus (living attenuated vaccine) as for example ,when using a defective syringe ,accidentally sprayed some vaccine in the eye as a result those veterinarians had contact the infection and developed acute brucellosis .

h-contact infections constitutes about from 60-70% of cases among veterinarians , butchers , farmers .

3-air borne infection include :
a-in and around the infected farms on the inhalation of dust of dried animals tissues and excreta
b-the minimum infective dose for humans by the way of the respiratory passages seems to be small.

4-man to man transmission :

It is very rare ,however ,a case was reported after blood transfusion ,and another case in achild from his mother .

Susceptibility : includes :

1-age 2-sex 3-occupation 4-season

1-age :

a-most frequently affected are in between 6-40 years

2-sex:

Males are infected more frequently than females (ratio 3:1) ,simply because dairy workers and meat handlers in slaughtered houses are mostly males .

3-occupation :

The disease is commonly confined to veterinarians ,farm workers or farmers (handling milk products as well as diseased animals) ,slaughter houses workers and laboratory workers .

4-season:

The disease is most prevalent in summer .these is an elevation of cases in april and reaching it is maximum in july and august

Sequelae of infection :

a-in apparent (latent infection)infection b- manifest disease

incubation period :

varies ,usually from 5-21 days ,but some times ,may be several months .it is difficult to ascertain ,onest is insidious and date of infection usually not established .

the disease in man :

a-the disease is a septicemic with sudden or insidious onest
b-is accompanied by continued , intermittened or irregular fever
c-the fever is prolonged with frequent remissions in which fever persists for 10-14 days followed by to 2-3 weeks a febrile period d-the symptomatology of the acute brucellosis ,like that of any other febrile diseases ,includes chills ,profuse sweeting and elevation of temperature .
f-an almost constant symptoms is weakness
g-any exercise produces pronounced fatigue
h-the temperature can vary from normal in the morning to 40 celicius degree in the afternoon .
i-sweeting is characterized by apeculair odour occurs at night

j-common symptoms are insomania ,sexual impotence , constipation,anorexia ,headache ,muscular and articular pains ,and general malaise .
k-this disease has amarked effect on the nervous system evidenced by by irritation ,nervousness and depressions.
l- many patients have enlarged peripheral lymph nodes or splenomegaly and often hepatomegaly ,but rarely jaundice.
m-brucella organisms localizes intracellulary in tissues of reticuloendothelial system,such as lymph nodes ,bone marrow ,spleen and liver .
n-tissues reactions are granulomatous .
o-the duration of disease can vary from aseveral few weeks or months to several years.

What are the serious complications of humans brucellosis $$$!!!!!

1-encephalitis 2- meningitis 3- peripheral neuritis 4-sponylitis 5-suppurative arthritis 6-endocarditis .

(my first clinical practical diseased case alone in a farm)

p-the chronic form of the disease occurs in some patients and may last for many years with or without the prescence of localized foci of infections . the symptoms are associated with hypersensitivity .

q-few cases of abortion in women and orchitis in man have been reported

r-persistence of infection associated either with acute relapses or chronic disease .

HISTORY OF BRUCELLA :

1-brucella melitensis discovered in island of malta in 1887 by bruce ,where a lot of soldiers died when they have drunk milk from goats ((malta fever)).

2-when the soldiers were forbidden to drink raw milk ,the disease disappeared .

3-the disease has wide distribution especially in Mediterranean countries ((Mediterranean fever)) .

4-the first case of undulant fever were reported in Egypt in by the ministry of public health in 1929.

DIAGNOSIS :

1-in man aclinical diagnosis of brucellosis based on symptoms and history should always be confirmed in the laboratory .
2-isolation and typing of casual agent is definitive and more over may indicate the source of infection
3-blood taken while the Patients is febrile is cultured on the appropriated media
4-sampling can also be taken from:
a-lymph nodes b-cerebrospinal fluids c-abscesses
5-it is recommended that the cultures be repeated several times especially in enzootic areas of brucella abortus .
6-due to the wide spread of usage of antibiotics before diagnosis in febrile patients ,bacteriologic examination often yield negative results and serologic tests become increasingly necessary.
Spread of disease in man in endemic areas :
1-sporadic cases and occasionally outbreaks (are you insists)
2-brucellin test is an intra dermaly hypersensitivity test which can be applied in survey studies to show survey studies to show prevalence of infection in man :
a-positive reaction means : past infections.
b-the intradermal test with non cellular allergens is useful for epidemiologic studies but not for clinical diagnosis.

Prevention and control :

The most rational approach for preventing human brucellosis is the control and eradication of the infection in animals reservoirs.

Measures for animals :

1-to prevent spread of infection by :
a-sanitary clean animal environment .
b-veterinary care
c-management of aborted animals and sanitary disposal of wastes.
d-in regions or countries with alow prevelance of bovine brucellosis (up to 15 %) what the excellents works :
a-an eradication program can be carried out by repeated serologic diagnostic tests applied to the entire herd
b- elimination of reactors until all foci of infection have been disappeared .
c- this procedures can be used alone in countries with alow prevalence or in combinations with the vaccinations of calves if you agood intelligences scanners.
d-epidemiologic surveillance and control and control of animal transport are very important in a such program .
e-vaccination is recommended for control of bovine brucellosis in enzootic areas with high prevalence rates (over 15 %),the vaccine of choice is brucella strain 19 confirmed by its world wide use ,the protection it gives for the useful life time of the animals and of low cost.

What are the advantages of vaccinations of brucella abortus strain 19 :

1-world wide use. 2-low cost.3-the protections it gives for the useful lifetime of the animals.

4-to avoid interference with the diagnoses it is recommended that the vaccinations be limited to young animals their ages between (calves of 3-4 months) .(why)!!!!because those animals rapidly lose the antibodies produced in response to the vaccines (memory cells eternal) .

5-it is estimated that 65% to 85% of vaccinated animals derive long term protection against infections.

6-the anti abortive effect of the vaccine is pronounced ,thus reducing one of the principle sources of the infections .

7-male calves and females over 8 months of age should not be vaccinated .

8-where possible , this upper limit should be 6 months.

9-revaccination is not recommended .

10-the objectives of a systematic and compulsory program of vaccination of calves in a given area or countery is to reduce the infection rate and obtain herds resistance to brucellosis.

11-so that , eradication of the disease may then begin .

12-it is estimated that 7-10 years of systematic vaccination are necessary to achieve this objective.

13-untill a few years ago the vaccination of adult cows with strain 19 was in advisable ,because of the prolonged persistence of antibodies that could interfere with diagnosis ,

14-several researchers proved that vaccination of adult animals with a smaller could impart an immunity comparable to that of a full dose ,while at same time agglutination titers stayed lower and disappeared faster.
15-complementary tests were very useful in distinguishing between reactions due to infection and those due to vaccinations.(by repeated serologic tests ,if the titer is increases it is an infection and if not or become lower it is vaccination (are you sure).
16-vaccinations of adult females may be considered in herds suffering acute brucellosis characterized by abortions and rapidely spreading infections and also as well as in large herds where chronic brucellosis has proven hard to eradicate .
17-the recommended dose is one to three billions cells of strain 19 brucella administered subcutaneously .
18-only animals testing should be vaccinated and they should be indelibly marked under government supervision.
19-at the beginning of the operation ,reactors should be eliminated immediately .
20-vaccinated animals should be examined serological 6 months later using rose Bengal ,mercapto ethanol and standard tube agglutination test .
21-these tests used also in diagnosing infections .
22-rose Bengal is screening test then standered tube agglutination test followed by mercapto ethanol test and those that have become infected should be send to slaughter .
23-using periodic serologic examination ,it is estimated that a problem herd can be free from infection in two years.
23-oral vaccination with strain 19 brucella is anew possibility (are sure from molecular biosynthesis) .

24-control of the infection caused by b.melitensis in goats and sheep is based mainly on vaccinations .
25-the preferred vaccine is brucella melitensis rev – 1 ,which is administered to 3-6 month old females.
26-the adult females animals can receive asmaller dose of the same vaccine (20,000 times fewer bacterial cells than the dose for young females).
27-how ever the control procedures of diagnosis and sacrificing reactor animals has produced satisfactory results in areas of low prevalence .
28-the control of swine brucellosis ,depends on test and slaughter policy as amean of eradications , no vaccines available for swines .
29-brucellosis caused by brucella canis in dogs kennels can be controlled by repeated serologic tests and blood cultures ,followed by eleminations of reactor animals .no vaccines are yet available .stray dogs must be eliminating.

Measures for man :

1-prevention of ingestion infections 2-prevention of occupational contact infections 3-prevention of air borne infections

Treatment :

1-treatment of infected persons by using :

a-tetracycline ,streptomycin , vitamin complex ,high protein diets , bed rest ,for at least three weeks .

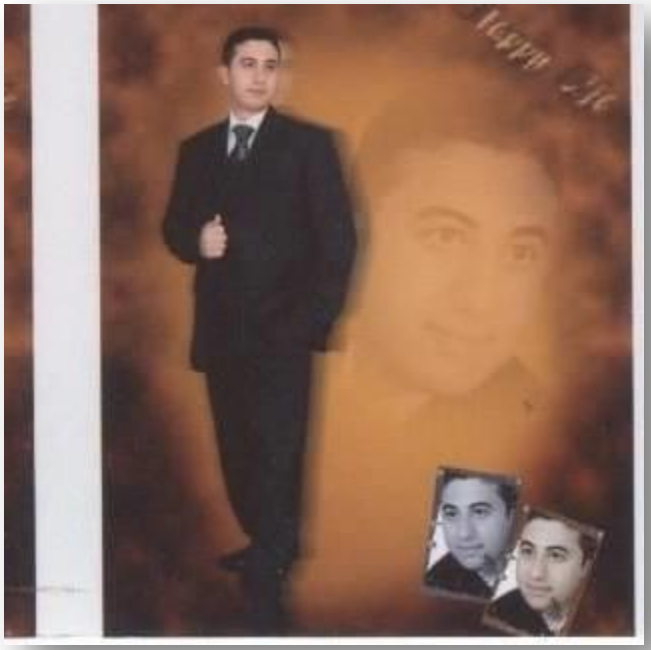
b-cortisone in the first three days of treatment.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA



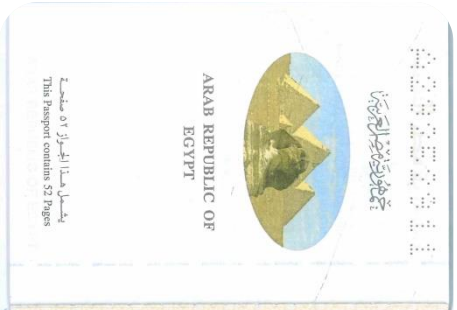
Bacteria

Bacteria, also called germs, are microscopic organisms not visible with the naked eye. Bacteria are everywhere, both inside and outside of your body.

Bacteria are small single-celled organisms. Bacteria are found almost everywhere on Earth and are vital to the planet's ecosystems. Some species can live under extreme conditions of temperature and pressure. The human body is full of bacteria, and in fact is estimated to contain more bacterial cells than human cells.

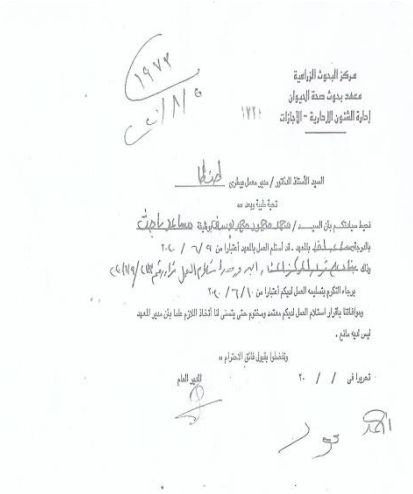
Bacteria are mostly free-living organisms often consisting of one biological cell. They constitute a large domain of prokaryotic microorganisms. Typically a few micrometres in length, bacteria were among the first life forms to appear on Earth, and are present in most of its habitats.

Most bacteria in the body are harmless, and some are even helpful. A relatively small number of species cause disease.


ARAB REPUBLIC OF EGYPT
P EGY A29252911
MOHAMMAD MAHMOUD MOHAMMAD YOUSSEF WAHIBA
09/09/1981 BENEIRA
EGYPTIAN M
10/11/2026
VETERINARIAN
P<EGYWAHIBA<<MOHAMMAD<MAHMOUD<MOHAMMAD<YOUSS
A292529113EGY8109091M2811109<<<<<<<<<<<<<<DO

جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتاريخ : ١٩٨١/٩/٩
الجنسية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٠٢١/٤/٢٧
تاريخ اعتماد مجلس الجامعة : ٢٠٢١/٥/١٩
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بقرار رقم ١٦٩ بتاريخ ٢٠٢٠/١٠/٥
المختص رئيس القسم وكيل الكلية للدراسات العليا عميد الكلية
مستخرج من بيانات مركز الشهادات الموحدة بتاريخ ٢٠٢١/٦/٢٠

جامعة
كفر الشيخ
شهادة تخرج
063342
تشهد كلية الطب البيطري
أن / محمد محمود محمد يوسف
المولود فى البحيرة بتاريخ ١٩٨١م
قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية
فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً
مجموع تراكمى ٢٤٧٥ / ٣٣٠٠ النسبة المئوية ٧٥,٠٠%
وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م
ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا
الموظف المسئول المراجع مدير شئون الطلاب عميد الكلية
تحريراً فى ٢٠١٢/٥/٢٢م


مركز البحوث الزراعية
معمل بحوث صحة الحيوان
إجازة الشئون الإدارية - الإجازات
السيد الأستاذ الدكتور / مدير معمل بيطرى
نحية طيبة وبعد ،،
يرجاء التكرم بتشابه العمل لديكم اختيارا من ٢٠ / ٦ / ٢٠
وبرفائنا بالقرار استلام العمل لديكم منتدبا وبستحوم حتى يتسنى لنا أخذذ القرار فضا بان مدير العديد
ليس لديه مانع .
وتفضلوا بقبول فائق الاحترام ،،
تحريراً فى / / ٢٠
المدير العام

جمهورية مصر العربية
بطاقة تحقيق الشخصية
محمد
محمود محمد يوسف وهيبه
ش العوض -كفر الزيات
مركز كفر الزيات - الغربية
٢٨١٠٩٠٩١٨٠٠٥١٦
HQ2270868

٢٨١٠٩٠٩١٨٠٠٥١٦ ٢٠١٨/٠٧
طبيب بيطرى
معهد بحوث صحة الحيوان بطنطا
ذكر مسلم أعزب
البطاقة سارية حتى ٢٠٢٥/٠٧/٢٣
٣

وحده مرور دمنهور
رخصه قياده خاصه
١١٣٦٢٢
جمهورية مصر العربية
وزارة الداخلية
مرور البحيره
محمد محمود
محمد يوسف
مصرى
بكالوريوس طب بيطرى
ش طور سيناء ش الشيخة
دمنهور.

Morphology

Bacteria are complex and highly variable microbes. They come in four basic shapes: spherical (cocci), rod-shaped (bacilli), arc-shaped (vibrio), and spiral (spirochete)

Cell wall

Eubacteria are enclosed by a cell wall. The wall is made of cross-linked chains of peptidoglycan, a polymer that combines both amino acid and sugar chains. ... Some types of bacteria have an additional layer outside the cell wall. This layer is made from lipopolysaccharide (LPS), a combination of lipids and sugars.

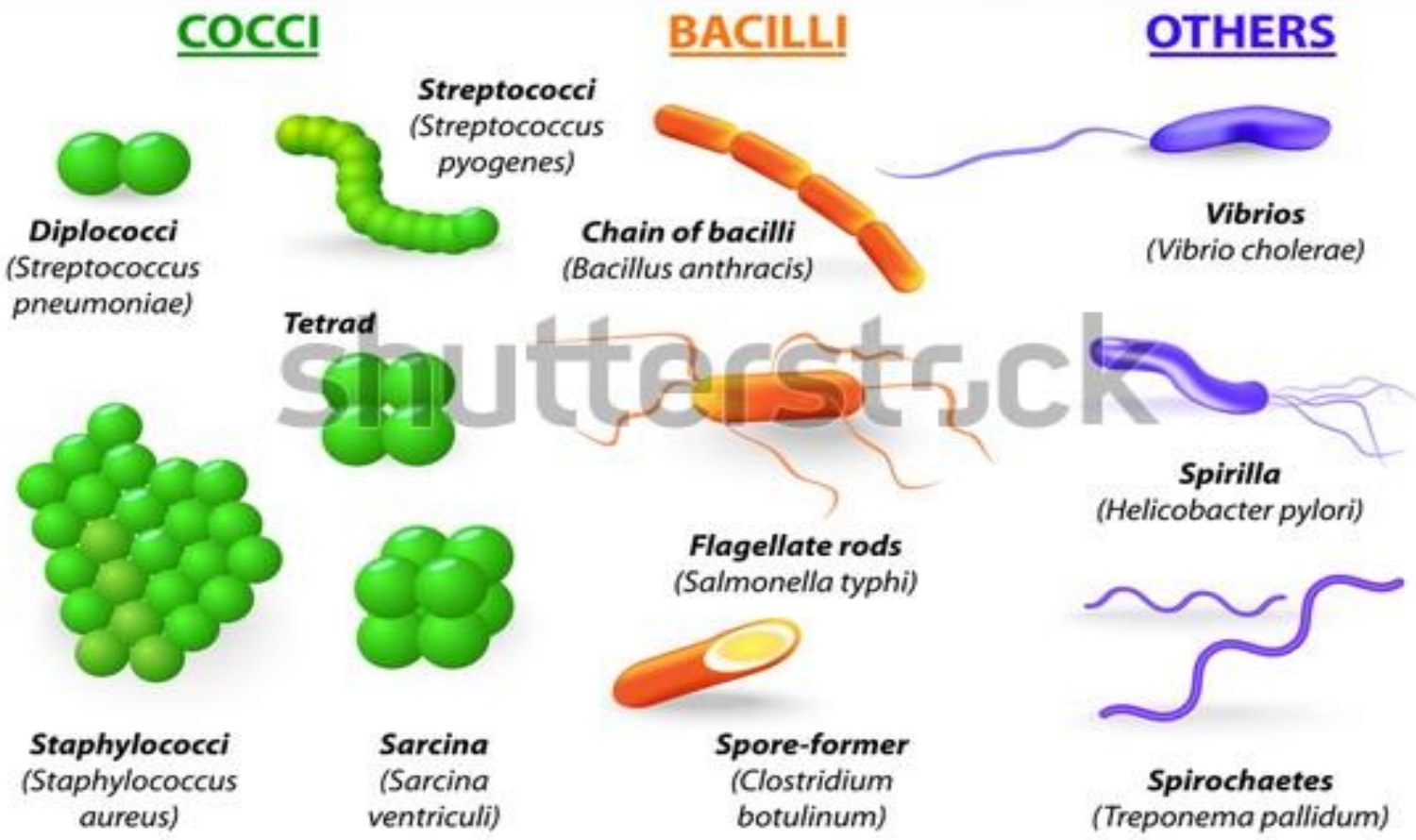
Nutrition

Nutrition. A great many of the most familiar eubacteria are heterotrophs, meaning they must take food in from outside sources. Of the heterotrophs, the majority are saprophytes, which consume dead material, or parasites, which live on or within another organism at the host's expense.

Treatment

Antibiotics are the usual treatment. When you take antibiotics, follow the directions carefully. Each time you take antibiotics, you increase the chances that bacteria in your body will learn to resist them causing antibiotic resistance. Later, you could get or spread an infection that those antibiotics cannot cure.

SHAPES OF BACTERIA



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SHAPES OF BACTERIA

SPHERICAL (COCCI)

Streptococcus pneumoniae

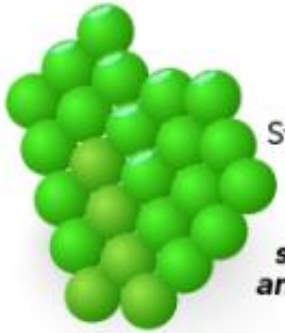
causes Pneumonia

Streptococcus pyogenes

causes Strep throat

Micrococcus luteus

causes armpits to stink



Staphylococcus aureus

can cause sinus infections and food poisoing

ROD-SHAPED (BACILLI)

(Bacillus anthracis)

causes Anthrax

(Salmonella enterica)

causes Typhoid

(Clostridium botulinum)

causes Botulism

SPIRAL-SHAPED (AND OTHERS)

(Vibrio cholerae)

causes Cholera

(Helicobacter pylori)

can cause stomach ulcers

(Treponema pallidum)

causes Syphilis

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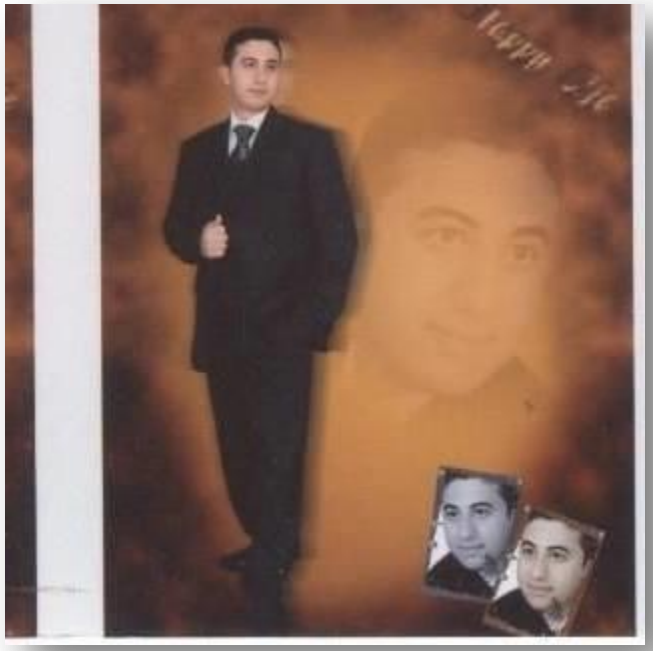
BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA



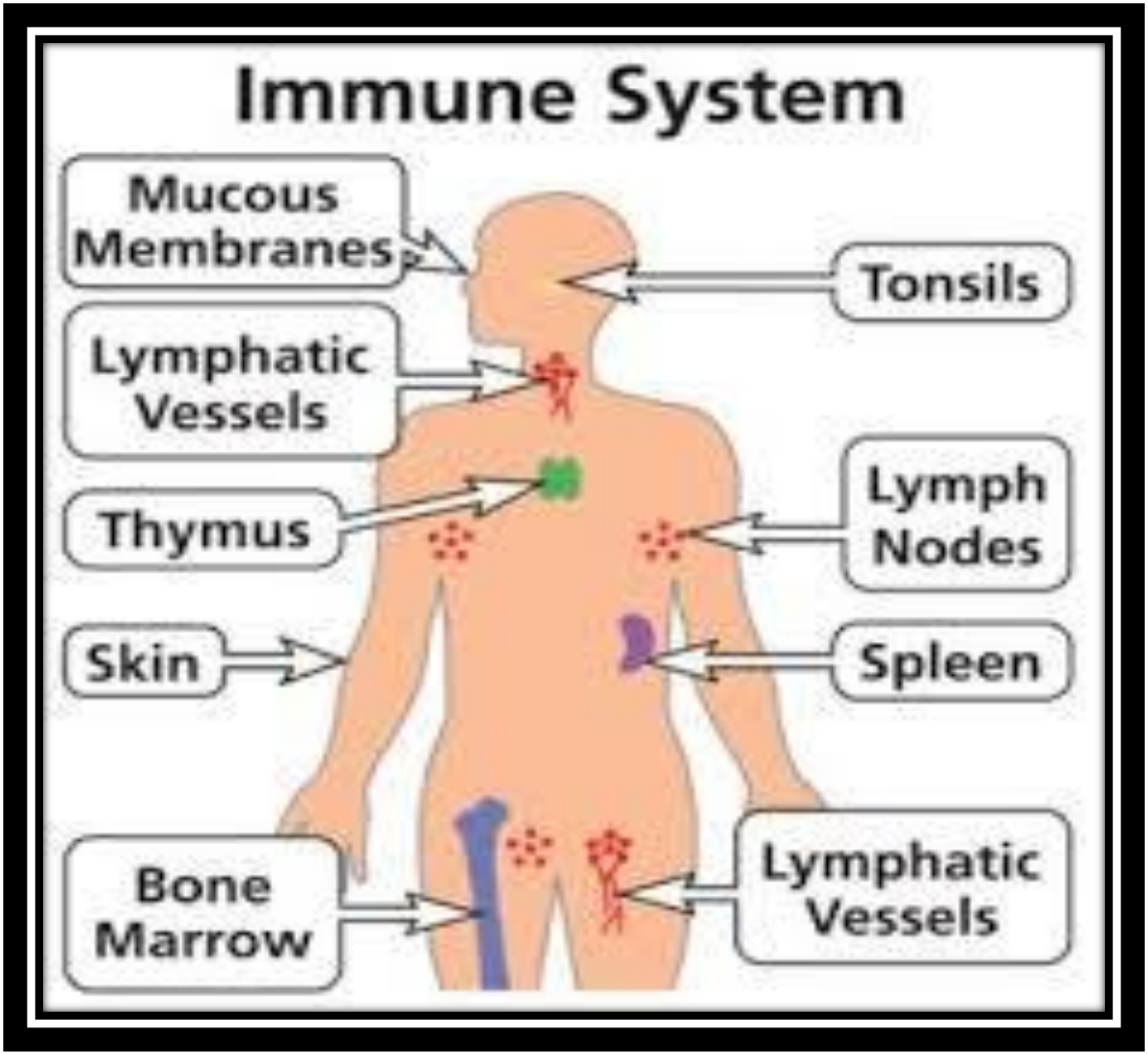
immune system

What is immune system and its function

Your immune system is a large network of organs, white blood cells, proteins (antibodies) and chemicals. This system works together to protect you from foreign invaders (bacteria, viruses, parasites, and fungi) that cause infection, illness and disease.

the organs and processes of the body that provide resistance to infection and toxins. Organs include the thymus, bone marrow, and lymph nodes.

What is the immune system? The immune system protects your child's body from outside invaders. These include germs such as bacteria, viruses, and fungi, and toxins (chemicals made by microbes). The immune system is made up of different organs, cells, and proteins that work together.


Immune System
Mucous Membranes
Tonsils
Lymphatic Vessels
Thymus
Lymph Nodes
Skin
Spleen
Bone Marrow
Lymphatic Vessels

What are the 4 immune systems?

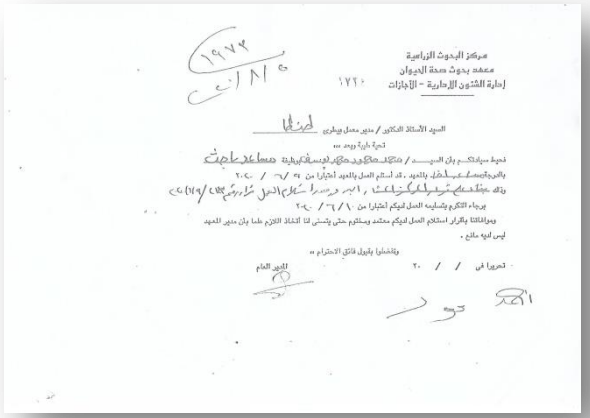
What are the 4 immune systems?
The main parts of the immune system are: white blood cells, antibodies, the complement system, the lymphatic system, the spleen, the thymus, and the bone marrow. These are the parts of your immune system that actively fight infection.

There are 2 main parts of the immune system:

1-The innate immune system. You are born with this.

2-The adaptive immune system. You develop this when your body is exposed to microbes or chemicals released by microbes.

These 2 immune systems work together.



The innate immune system

This is your child's rapid response system. It is the first to respond when it finds an invader. It is made up of the skin, the eye's cornea, and the mucous membrane that lines the respiratory, gastrointestinal, and genitourinary tracts. These all create physical barriers to help protect your child's body. They protect against harmful germs, parasites (such as worms), or cells (such as cancer). The innate immune system is inherited. It is active from the moment your child is born. When this system recognizes an invader, it goes into action right away. The cells of this immune system surround and cover the invader. The invader is killed inside the immune system cells (called phagocytes).

The acquired immune system

The acquired immune system, with help from the innate system, makes special proteins (called antibodies) to protect your body from a specific invader. These antibodies are developed by cells called B lymphocytes after the body has been exposed to the invader. The antibodies stay in your child's body. It can take several days for antibodies to form. But after the first exposure, the immune system will recognize the invader and defend against it. The acquired immune system changes during your child's life. Immunizations train your child's immune system to make antibodies to protect them from harmful diseases.

The cells of both parts of the immune system are made in different organs of the body, including:

Adenoids. Two glands located at the back of the nasal passage.
Bone marrow. The soft, spongy tissue found in bone cavities.
Lymph nodes. Small organs shaped like beans, which are located all over the body and connect via the lymphatic vessels.
Lymphatic vessels. A network of channels all over the body that carries lymphocytes to the lymphoid organs and bloodstream.
Peyer patches. Lymphoid tissue in the small intestine.
Spleen. A fist-sized organ located in the belly (abdominal) cavity.
Thymus. Two lobes that join in front of the windpipe (trachea) behind the breastbone.
Tonsils. Two oval masses in the back of the throat.

القرآن الكريم مخطوطا.pdf

أقسام البكتيريا = +والطب الشرعى = +وصحة الأغذية = +والباثولوجيا.pdf

RECENT DISCUSSIONS MATERIALS FOR RESEARCHER ASSISTANT MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA 28109091800516-20045788019 66.pdf

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

بعض التحصينات الإجبارية للأمراض الوبائية والكشف عنها داخل القطر المصرى
(كتبها ونسقها
دمحمدمحمودمحمديوسف وهيبه
بعد إرسالها المركزى) -

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

المرض المشتبه به	نوع العينة المراد العزل منها	المادة الحافظة	طريقة النقل المناسبة ودرجة الحرارة المطلوبة	المدة المسموح بها حتى الوصول إلى معهد الدقى
١-الحمى القلاعية (FMD)	حويصلة كاملة-بثرات- سائل الحويصلة	Glycerin buffer	فى ثلاجة نقل العينات محتوية على أكياس ثلج خاصة (4C) بحيث تكون كل عينة فى كيس منفصل	٢٤ ساعة
٢-حمى الوادى المتصدع (RFV)	دم على مانع تجلط –أجنة مجهضة (كبد-طحال-غدة ليمفاوية)	Universal transport medium	كما سبق	٢٤ ساعة
٣-الجلد العقدى (LSD)	عقدة جلدية – عقدة ليمفاوية – طحال	Transport buffer	كما سبق	٢٤ ساعة
٤-طاعون المجترات الصغيرة (PPR)	دم على مانع تجلط أثناء إرتفاع درجة الحرارة –بثرات من الفم – مسحات من العين والأنف والفم – أحشاء (الرئة غدد ليمفاوية – الأمعاء الطحال)	EDTA Heparin	كما سبق	٤-٦ساعات

٥-التسمم الدموى Pasteurella multocida	مسحات أنفية – أحشاء داخلية – دم –سيرم		كماسبق	٤-٦ساعات
٦-حمى الثلاث أيام Bovine ephemeral fever virus Rhabdoviridae	دم على مانع تجلط – غدة ليمفاوية - طحال	Preservative media PBS	كما سبق	٢٤ ساعة

٧- الإلتهاب الشعبى البقرى (IBR)	مسحات أنفية-غدة ليمفاوية-جزء من الرئة – جزء من القصبة الهوائية	Preservative media PBS	كما سبق	٢٤ساعة
٨-الإسهال المعدى (BVD)	مسحات أنفية-مسحات شرجية-سيرم-غدة ليمفاوية – أحشاءداخلية	Preservative media PBS	كما سبق	٢٤ساعة
٩-أمراض العجول الرضيعة pneumoenteritis,parainfleunza ,corona viruses,may be mixed with salmonella , mycoplasma, E,coli	عينات براز-سيرم-أحشاءداخلية-مسحات أنفية ودم	Preservative media PBS	كما سبق	٢٤ساعة

١٠-التسمم المعوى (Enterotoxaemia) Cholesteridial infections especially in sheep and goat farms need co vac 8	الأمعاءالدقيقة (مربوطة ولا يقل طولها عن ١٥ سم) –فص كامل من الكبد به الإصابة – كلية كاملة		كما سبق	٤-٦ساعات
١١-التفحم العضلى والأوديما الخبيثة Black Leg (black quarter) Malignant Edema Closteridium Choviae	العضلة المصابة المتفحمة – سائل إرتشاحى من مكان الإصابة		كما سبق	٤-٦ساعات
١٢-المرض الأسود Black Closteridium (Disease) Choviae	فص كامل من الكبد به إصابة –الأمعاء الدقيقة		كما سبق	٤-٦ساعات

١٣-باسيلارى هيموجلوبين يوريا (بول أحمر) (Bacillary Hemoglobinuria) Clostridium novyi type D	فص كامل من الكبد به الإصابة		كما سبق	٤-٦ساعات
١٤-تعفن الحافر (Foot Rot) Fusobacterium necrophorum	مسحات من الحافر المصاب بعد تنظيفه جيدا من الأنسجة الميتة	المسحات على ناقل (Cary-Blair Medium)	كما سبق	٤-٦ساعات
١٥- التسمم الغذائى	-الأعلاف (لا تقل العينة عن ٥٠-١٠٠جرام) -السيلاج (لا تقل العينة عن ٥٠-١٠٠ جرام)- المياة (لا تقل عن ٢٠٠ مل)		كما سبق	لا تزيد عن ١٢ ساعة بطريقة الحفظ المذكورة

ملحوظة هامة : البروسيلاوالإسبريجلوسيس يسببان عاصفة من الإجهاضات ومن الأمراض التى يجب تبليغ جميع السلطات الأمنية الغذائية ويكتب ويرفع بها تقرير مبين وتم نسيانها من المختصين العاليين

يجب الإفادة عن الموقف التحصينى لتلك الأمراض عند إرسال أى عينات للفحص

وكيل المعهد للتشخيص المعملى وصحة الأغذية

تحريرا فى : / /

يعتمد

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RECENT DISCUSSIONS MATERIALS FOR RESEARCHER ASSISTANT MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA 28109091800516-20045788019 66.pdf

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

جامعة بنها

كلية الطب البيطرى

قسم البكتيريا والمناعة والفطريات

كتبهاونسقها مساعدباحث محمدمحمودمحمديوسف وهيبه

ماجيستير البكتيريا والمناعة والفطريات

معهد بحوث الصحة الحيوانية

مركز البحوث الزراعية

وزارة الزراعة

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS

FAMILY: ENTEROBACTERIACEAE

THE DEFINITION OF ENTEROBACTERIACEA:

Intestinal bacteria describe large biochemically and antigenically related groups of organisms

FAMILY: ENTEROBACTERIACEAE

1-Some of them are pathogenic potential to man, animals, insects and plants.

2- It contains about 4 tribes, 12 genera on the basis of DNA relatedness.

3-Generally the members of family enterobacteriaceae is composed of gram negative rods which may be motile by peritrichous flagella or non-motile.

4- They are non -spore forming and not acid fast.

5- Most of them are non capsulated but some have capsules or microcapsules (slime layer).

6- Enterobacteriaceae members grow on the presence and the absence of free oxygen because facultative anaerobes have the presence of respiratory and fermentative metabolisms.

5- They grow well on ordinary media.

6- They are similar in the morphological and cultural characters.

7-Moreover all species contain the enterobacterial common antigen.

8- Biochemically they produce acid and gas due to the fermentation of glucose or different types of carbohydrates.

9- All species are catalase positive .

10-Reduce nitrate to nitrite.

11- Oxidase negative .

12- According to their ability to ferment lactose they were differentiated biochemically into two divisions :

1 - Lactose fermenting enterobacterial genera.

2-Non lactose fermenting enterobacterial genera .

This classification depends on the cultivation of the species of family enterobacteriaceae on one of the following differential selective media :

1-MacConkey agar (lactose-bile neutral red agar)

2-S.S media (salmonella- shigella agar)

3- Desoxycholate cirate agar

4- Hegkton media

Different genera of Enterobacteriaceae according to their ability to ferment lactose : Lactose fermenter and Non Lactose +(Reacted Fundamental Environment) fermenter

Lactose fermenter :

Genus	Habitate
E.coli	Intestine
Enteobacter	Intestine , water
Citrobacter	Intestine, water , soil
Yersenia	Intestine, water
Serratia	Intestine, water , ,soil

Non- lactose fermenter :

Genus	Habitate
Shigella	Intestine ,water, soil
Salmonella	Intestine , water , soil
Hafnia	Intestine, water,soil
Proteus	Intestine, water , soil
Morganella	As Proteus

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

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Agricultural Research Center

.THANK YOU .

WORLD MAP
1. Netherlands
2. Belgium
3. Luxembourg
4. Switzerland
5. Slovenia
6. Croatia
7. Bosnia and Herzegovina
8. Czechia
9. Slovakia
10. Austria
11. Hungary
12. Serbia
13. Moldova
14. North Macedonia
15. Albania
16. Cyprus
17. Lebanon
18. Guinea-Bissau
19. Guinea
20. Ghana
21. Togo
22. Benin
23. Cameroon
24. Equatorial Guinea
25. Rwanda
26. Cambodia
27. Panama
28. Malawi
29. Liechtenstein
30. Montenegro
31. Kosovo
32. Palestinian Territories
33. St. Vincent & the Grenadines
0 2,000 4,000 Miles
0 2,000 4,000 6,000 8,000 Kilometers
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BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

GENUS: ESHCERICHIA
SPECIES: ESCERICHIA COLI (E.Coli)
Synonym: BACTERIUM COLI (E.Coli)

Definition: Escherichia coli is a normal inhabitant of the lower part of intestine of all warm blooded animals.

It is rarely present in stomach and in few numbers in the anterior part of intestine. Carnivores and omnivores usually harbor E.coli in greater number than herbivore.

Under certain conditions of stress and environmental factors may undergo to be potential pathogenic causing sever diseased conditions.

Specific diseases caused by enteropathogenic E.coli in different animal species:

Species	Disease	Symptoms
Childrens	Coli septicemia	**Diarrhea , death**
Newly born animals	Colibacillosis, white or calf scour	**Septicemia and rapid death**

Microscopic characters :

E.coli is coccobacilli to long rods (.6 x1.3M), arranged singly or in short chains especially in mucoid colonies where it had a slime layer of polysaccharides . it is motile and non – sporulated .

Cultural characters :

(Nutrient broth-Nutrient agar-Blood agar-MacConkey agar)

-In nutrient broth growth is rapid , gives regular turbidity within 12-18 hours at 37 C with heavy powdery sediment after 24 hours .

-On nutrient agar growth

1-smooth surface colonies occurs after 24 hours,

-The colonies are grayish in color , circular , convex 2-3mm in diameter .

 2- mucoid colonies appear due to the presence of slime layer and larger in size than smooth one .

-On blood agar the surface colonies are larger some are hemolytic and others are non-hemolytic

-On macConkey agar it gives round pink colonies, medium sized opaque colonies i.e lactose fermenter

(Viability And Resistance) :

-It may survive for weeks or months in contaminated water, dwellings in the animal houses in the presence of mucous or feces . Some strains resist dryness.

-It is susceptible to physical and chemical agents. E.coli is killed by heating at (55-60 C) for 30 minutes, within 10 minutes by autoclaving.

The Antigenic Structures Of E.coli include three antigens : 1-somatic antigens 2- capsular antigens 3- flagellar antigens . Detected by serological diagnosis

1-Somatic antigens (146 O- antigens)
2-Capsular antigens (91 K –antigens)
3- Flagellar antigens (49 H- antigens) :

1-Somatic Antigens : (146 O- antigens) :

E. coli strains have 146 O-antigens in their body . Each strain carries only one type of O-antigen . They are composed chemically of polysaccharide–phospholipids –protein complex. The O –antigens are designated by numbers as O1, O2, O3,O4 ,O5 – O 145 etc .

The O – antigens are heat stables.

2- Capsular antigens (91 K –antigens) :

These 91 k-antigens occur as a slime layer or microcapsule on most enteropathogenic E.coli strains.

Chemically they are composed of poly saccharides . k-antigens prevent agglutination between O-antigens and their specific homologous antisera.

The K – antigens are differentiated into

3 types according to their thermo stability as follows :

L-antigen : heat labile

A- antigen : heat stable

B- antigen : lipopoly saccharide destroyed by autoclaving

The K-antigen is very important in serotyping of E.coli as well as in the mechanism of pathogenesis as K 88 and K 99 .

These K 88 and K 99 mediate adhesion of the organism to the microvilli of the intestinal epithelial cells and enhance colonization .

3-Flagellar antigen (49 H-antigens) : There are 49 H-antigens which is composed of protein . frequently it is poorly developed .

It has weak role in serotyping of E.coli.

-Serotyping Identification :

For serotyping of E. coli , identification of O and K antigens is applied by slide agglutination test to determine its antigenic structures as O20/B6 , O55/B5 , O88/B4 , O111/B4 .

Toxins of E.coli (E.coli products) :

1-Enterotoxin 2- colicins

1-Enterotoxin:

This substances is produced by entero pathogenic strains of E. coli , and causes diarrhea in man and animals . Injection of E .coli enterotoxins into the ligated intestinal loops of rabbits induced swelling , oedema and accumulation of exudate in the injected loop. The enterotoxins are not produced by all E.coli strains but only the enteropathogenic.

E.coli enterotoxin is composed of two fractions:

a- Heat stable fraction.
b-Heat labile fraction.

a-Heat stable fraction : it is exteracellular in the culture . it is resistant to heating for 60 C but destroyed by autoclaving .
b- Heat labile fraction: it is inactivating by heating at 60 C for 30 minutes it is antigenic and occurred within bacterial cells . it could be neutralized by specific antisera .

2- Colicins :

It is E.coli products and composed chemically of protien . it has bactericidal activitiy . colicins productions is very important in differentiation of E.coli strains into :

Colicins positive E.coli.

Colicins negative E.coli.

Laboratory diagnosis:

- The isolation of E.coli may be of no value as E.coli are normal inhabitant of the intestinal tract, unless other seven factors are considered including:

1-Detailed history of the case, symptoms and lesions with differential bacteriological diagnosis on the other causative agents of digestive disorders .

2-Purity of the growth of E.coli on the primary cultures note the growth of lactose fermenting colonies on MacConkey agar .

3-Site of isolation from various organs especially from the parenchymatous organs as liver , spleen , mesenteric lymph nodes and lung tissues indicating colibacillosis.

4- Biochemically identification of isolated suspected E.coli the suspected E.coli colonies as lactose fermenter on macConkey media can be tested biochemically And must be differentiated from the other lactose – fermenter species by the following reactions (Indol , Methyle Red , VP , Citrate , Urea Hydrolysis , H2S, Motility , KCN) (Voges proskauer)(potassium cyanide)

E.coli are motile and Indol and Methyl Red Positives Only .

5-Serotyping of E. coli if the isolating colonies, are biochemically typical of E.coli, Serotyping must be performed using polyvalent O, K anti-sera to determine the antigenic structure of the isolated strain of E.coli.

6-Final diagnosis depends mainly upon the isolation of E.coli from various fresh organs in case of septicemia of the same antigenic structure.

7-It is very essential that the same serotype of E.coli must be isolated from feces or intestinal content as well as the internal organs of the same case .

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BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

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Occurrence of food borne pathogen in some types of soft cheese in local market

A Thesis Submitted to
Faculty of Veterinary Medicine
Benha University

By

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(BVSc., Kafer Elsheikh University, 2003) For the degree of M.Sc.
(Bacteriology, Immunology, Mycology)

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بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

(قالوا لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم)

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He works at animal health research institute, Tanta branch, Connected To The Main Institute In The Dokki -Giza -Cairo Under Super Visions Of Agricultural Researches Center Under Laws Of Universities Learning And Researches Qualifications.

Dedications

My Father
My Mother
My Sister
My Brother
My Uncles
My Family

Always all support religious forever.

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1. INTERODUCTION

Food safety is considered one of the most common urgent matters in the food industry worldwide. Spoilage of the food products with foodborne pathogens receive a special concern among food producers, investigators and customers. Consequently, producing a safe food represents one of the most imperative urgencies in the food processing (Friedman *et al.*, 2002; Mohamed *et al.*, 2013).

The outbreaks of foodborne illnesses caused by *E. coli* have been studied previously in developing countries after ingestion of milk products such as old-style cheese which is considered the major source of various types of pathogenic bacteria (Elhaida and Mohammed, 2013).

E. coli **is one of the major significant bacteria, which has a bad effect on both human and animal species. Thus, this type of bacteria can deteriorate the milk particularly raw milk and other milk products as a result of poor hygienic measures (Lara *et al.*, 2016; Garbage *et al.*, 2016).**

Prevention of foodborne illness is a global concern that affects all aspects of food production, including dairy. Dairy cattle are known reservoirs for zoonotic bacteria and milk and milk products have been implicated in outbreaks of foodborne illness (Wells *et al.,* 1991; Zhao *et al.,* 1995; Wallace, 1999). These outbreaks are generally caused by improperly pasteurized products, post pasteurization contamination, or by raw milk and raw milk products.

pathogenic *Escherichia coli* are also present in dairy production environments and have been associated with milk and milk products (Ryser, 2001; Watseka and Kinema, 2003). Although milk can be contaminated with bacterial pathogens via shedding in the udder, these zoonotic pathogens are not commonly associated with mastitis. Fecal contamination is considered to be the most common route of bulk tank milk contamination with these organisms.

Escherichia coli **live commensally in the gastrointestinal tract of most mammals, including humans, without causing disease. However, a small fraction of *E. coli* are human pathogens and have been implicated in foodborne illness with increasing frequency over the last 2 decades. Ingestion of enteropathogenic *E. coli* (EPEC) can result in a mild gastrointestinal disease that is relatively self-limiting but a subset of the EPEC, can a serious potentially fatal illness (O'Brien and Kipper, 1998). The most common human clinical EPEC isolated in the United States is O157:H7 (Canavalia *et al.,* 2001; Watseka and Kinema, 2003).**

E. coli **is classified into six pathotypes: enteroaggregative, enterohemorrhagic Shiga toxin-producing *E. coli* (STEC), enteron invasive, enteropathogenic, enterotoxigenic, and diffuse adherent (Jafari *et al.*, 2012). lethal STEC which cause severe illnesses were also detected (Boutin *et al.*, 2007).**

Previous studies indicated that STEC represents one of the most significant pathotypes which lead to foodborne illnesses compared with other types *E. coli* (Brett *et al.*, 2003; Kaufmann *et al.*, 2006). In human, the pathogenic effect of STEC nearly due to its ability to produce certain types of cytotoxins for example Shiga toxins (*stx1*), and intimin (*eae A*) virulent genes (Slaney *et al.*, 2009 and Assupol *et al.*, 2015).

Cheesemaking is a major industry worldwide, and much of it is still practiced on a relatively small scale with only basic hygiene practices (Kostas *et al.*, 2010; Dias *et al.*, 2012).

Among the dairy products, cheese is considered one of the most public sources of vital nutrients (*e.g.* vitamins, minerals and proteins) which represent the main part of healthy food (López-Expositor *et al.*, 2012). In Egypt. Moreover, Kariesh cheese is a prevalent type of cheese which contains a higher protein content with a small amount of fats (Hamad, 2015).

Nevertheless, cheese is considered a safe food for human being, sometimes its deterioration by various types of foodborne pathogens may take place. Enteropathogenic *E. coli* (EPEC) are considered the most common bacteria isolated from cheese. EPEC also cause high morbidity and mortality rates among young and old people (Kostas *et al.*, 2010).

Kariesh cheese are the most popular soft cheese in Egyptian cities and Arabian countries, similar to Domiati (Brown, 2004; Abd-Hamid, 2012; Hegazi *et al.,* 2012). It is an acid coagulated fresh cheese, made from skimmed buffalo's or cow's milk or a mixture of both with soft composition, white curd and slightly salty (Francois *et al.,* 2004; Abou-Donia , 2008), but mostly produced from skimmed buffaloes' milk and its manufacturing depends on acid coagulation by the action of rennet & lactic acid bacteria

Escherichia coli **is commonly present in the intestinal tracts of humans and animals, and its detection in processed foods is an indication of fecal matter contamination. It also indicates the inadequate controls of the environment of the cheeses processing units, the personals hygiene's of the food handlers, and the poor qualities of the finished products (Kostas *et al.*, 2010; Dias *et al.*, 2012).**

Because the occurrence of an Outbreaks of a foodborne disease is caused by Enteropathogenic *E. coli* and is associated with the consumptions of soft ripened cheese (Maurier *et al.*, 1973), the presence of these microorganisms in cheese has taken on a major significance.

Shiga toxins–producing *E. coli* (STEC), also known as serotoxins-producing *E. coli* (VTEC) or Enterohaemorrhagic *E. coli* (EHEC), have been known as a group of highly pathogenic *E. coli* strains producing one or more Shiga toxins (Monaghan *et al.*, 2011).

STEC represent a hazardous public health problem worldwide causing various human gastrointestinal tract diseases, including watery or bloody diarrhea and might develop a life-threatening disease, such as hemorrhagic colitis (HC), Thrombotic Thrombocytopenic Purpura (TTP) and Hemolytic Uremic Syndrome (HUS) (Pennington, 2010).

STEC strains produce two powerful phages encoded cytotoxins causing tissue damage in humans and animals, called Shiga toxins or serotoxins (*Stx1/VT1* and *Stx2/VT2*), which are the common features and main virulence factors of STEC and are directly correlated with human pathogenicity. (Lindgren *et al.*, 1993).

Stx1 **is one of the most powerful toxins, and the toxin producing strains are usually associated with more severe infections (Monies *et al.*, 2004 and Giles, 2007).**

STEC serotypes including O157:H7 STEC and non-O157. The O157:H7 STEC strain is considered one of the most important of all known food-borne pathogens because of the severity of associated illnesses produced by it and also, the apparent low infective dose (of less than 10 cells) (Bach *et al.*, 2002, Blanco *et al.*, 2003).

The most common, non-O157 strains associated with disease in humans including O26, O91, O103, O111, O128, O113 and O145 are responsible for up to 20 to 50% of all STEC infections (Johnson *et al.*, 2006).

There is a lack of information concerning the stress responses of many non

O157:H7 STEC causing human illness under stress conditions as most of these studies only focused on O157:H7 (Allen *et al*. 2008; Dong and Schell horn 2009; Vanaja *et al*. 2010). *eae* Agene it an intimin secretion gene that enhance attachment and colonization of *Escherichia coli* in the intestinal epithelium leading to sever cellular destruction and severe infections and sever symptoms like profuse diarrhea and ulcerative colitis (*Ibrahim et al.*, 2019*).*

Damiati cheese is one of soft pickled cheese and makes up about 75% of the cheese produced and consumed due to its nutritional value, convenience and have rich taste, strong sharp flavor as well as smooth body and texture.

Tallaga cheese is the most popular local type of packaged or unpackaged soft cheeses by all socioeconomic classes in Egypt due to its nutritional values, conveniences and good tastes, clean pleasant creamy low salty taste with a spreadable mellow soft body. It is a product closely related to Domiati cheese and mainly ready for consumption within one month of storage at refrigerator temperature.

Cheeses are more susceptible to spoilage due to microbial contamination. The source of contamination include both intrinsic factors (nutrients, water activity, PH, temperature, inhibiting factors produced by starter cultures and nonstarter microorganisms, (competitive microflora, etc.) and extrinsic factors (microbial quality of raw milk, production phases, ripening and packaging conditions, presence of chemical preservatives), cheese vat, cheese cloth and curd cutting knife, production room air and storage room(Hosni *et al.,*2011).

The improperly cleaned plants, equipment and food handlers are the main sources of contaminations to the cheese. Consumption of contaminated cheese with pathogenic microorganisms and their toxins can be used serious food borne problems to threat public health and big economic losses. But the most traditional cheeses are usually produced under poor hygienic conditions with different manufacturing technologies in local dairy plants.

***Escherichia coli* (*E. coli*) is one of normal inhabitant microorganisms of large intestine in human and warm-blooded animals. The main source of *E. coli* in raw milk and milk products is fecal contamination together with poor hygienic practices (Garbage *et al.*, 2016; Lara *et al.*, 2016). However, certain serotypes of *E. coli* have acquired some virulence-associated genes that enable them to cause intestinal or Extra-intestinal diseases (Hodson *et al.*, 2017).**

E. coli **is generally used as an indicator of direct or indirect fecal contamination and the possible presence of enteric pathogens in milk and dairy products (Kurnai and Johnson, 2001).**

These serotypes that cause enteric infections are generally called diarrheagenic *E. coli* strains, and their pathogenesis is associated with a number of virulence attributes, which vary according to pathotype.

Currently, diarrheagenic *E. coli* strains are classified into seven main pathotypes based on their distinct virulence determinants and pathogenic features, including enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohemorrhagic *E. coli* (EHEC), shiga toxin-producing *E. coli* (STEC), enter invasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAEC) and diffusively adherent *E. coli* (DAEC) , Uropathogenic *E. coli*

Uropathogenic *E. coli* (UPEC) is the most frequent pathogen responsible for up to 80% of UTIs have special virulence factors, including pili or fimbriae, especially type 1 which have the structural rule gene called *Fim H* gene which mediate attachment to uroepithelial and vaginal cells, resistance to human serum bactericidal activity, hemolysins production.

virulence factors of UPEC strains have a significant role in development of UTIs. The most virulence factor dependent upon the UPEC include adhesions type 1 fimbriae encoding by *Fim H* gene (Rutter *et al.*, 2006; Xia *et al.*, 2010) (Zahren et al 2014).

The serotypes associated with illness in humans include members of O serogroups 26, 91, 103, 111, 113, 121, 145 and 157 (Karbala *et al.*, 2010; Pizarro *et al.*, 2014).

As soft cheeses (Kariesh, Talega and Damietta) collected from different localities from Gharbia governorate may subjected to contaminations with *E. coli* and may constitute public hazard.

E. coli **constitutes publics hazards of living creatures especially humans (likes meningoencephalitis, pyelonephritis, hepatitis, pneumonitis, pleurisy, pericarditis, peritonitis, osteomyelitis, appendicitis).**

Therefore, this work was planned to covers the followings points:

1-Isolations, biochemicals and serologically identifications of pathologically pathotypes of Escherichia coli from Kariesh, Tillage and Damietta types of cheeses in el _Gharbia governorate.

2-Molecular identifications of isolated bacteria (Bacterium Coli) using PCR Techniques)

BACTERIAE

Lectures Written
And Presented By
Doctor
Mohammed Mahmoud Mohammed Yousef Wehaba
Stipulated Course Of Bacterial Genatics Arranged By Department Of Veterinary Bacteria,immunology ,Mycology to pass exams
department ,benha university

SOME GENATIC WORDS

1-dalton الذرة

2-nicked مكشوف

3-integrated اندماجى و موحد ومستكمل

4-off spring سلالة ونسل

5-inter breeding تهجين ومزج السلالات

6-alternatively إرادى و طوعى

7-um جزء من المليون من المتر

8-auto باد ئة

9- vector ناقل

10-supplementary اضافى - ملحق

11-temperate معتدل - غير متطرف

12-lytic انحلالى - انهيار

13-autonomously مستقل – إستقلالى

14-dissemination إنتشار

15-transduction

16-lysogenic مستذيب - مسبب للتحلل

17-prophage طور أولى - لاقم أولى

18-conversion تحويل

19-confer يحدث – محادثة - يحول

20-phenotypic النمط الظاهرى

21-progeny أنسال

22-competence إستحقاق –جدارة - أهلية –إجادة

23-homology علم الشباة – تشابه تركيبى - تماثل

24-defective معيبة - بها عيوب

25-complement متمم - مكمل

26-inversion إنعكاس - إستقلاب

27-duplication مضاعفة

28-extensive واسع النطاق

29-substitution إستبدال

30-nucleases هو إنزيم يعمل على تحلل الحمض النووى الريبوزى منقوص الأكسجين بواسطة فك الروابط الموجودة داخل النيوكليوتيدات المتجاورة

31-recombination إتحادات جديدة والتوليف الجينى وإعادة التركيب وعودة الإلتئام

BACTERIAL GENETICS

BACTERIAL GENATICS OF MEDICAL IMPORTANCE OF BACTERIAL CELL

1-VIRULENCE
2-PATHOGENICITY
3-ANTIBIOTIC RESISTANCE

DETERMINED BY GENETIC INFORMATION IN THE CELL GENOME

BACTERIAL GENETICS

: is the study of how genetic information is transferred, either from: a particular bacterium to its offspring or between interbreeding lines of bacteria, and how that genetic information is expressed

THE GENETIC INFORMATION IS NORMALLY ENCODED BY THE SPECIFIC SEQUENCE OF NITROGENOUS BASES

Adenine A Guanine G Thyamin T Cytosine

Gene • **A sequence of DNA carrying codon**

Codon • **The genetic information is stored**

WHAT IS DEFINITION OF A DNA MOLECULE :

1- A DNA IS COMPOSED OF TWO CHAINS OF NUCLEOTIDES WOUND TOGETHER IN THE FORM OF ADOUBLE HELIX

2- EACH CHAIN HAS ABACK BONE OF DEOXYRIBOSE AND PHOSPHATES RESIDUES ARRANGED ALTERNATIVELY

Sources of genetic information in bacteria :

1-Bacterial Chromosome

2-Plasmid

3-bacteriophages

4-Transposons

Bacterial Chromosome

- Bacterial nucleoid contains a circular chromosome of double stranded DNA.
- About 1000 um long when straightened.
- Prokaryotic cells are haploid " has single chromosome copy

Plasmid

- Circular extra-chromosomal DNA molecules, present in cytoplasm of bacteria.
- Capable of auto-replication.
- Can transfer genes from one cell to another.
- Act as Vectors in genetic engineering.
- Can also present in yeasts

Plasmid

❖ carry supplementary genetic information coding for beneficial properties as antibiotic resistance

- Not essential for normal growth and reproduction of Bacteria.
- but may confer survival advantages “ antibiotic resistance” , toxin production

Plasmid

- Plasmid can be integrated with chromosomal DNA.
- Episomes: integrated form of plasmid with DNA.

Plasmid can transfer from cell to cell by : 1- Transduction 2- Transformation 3- Conjugation

- **Widely used for cloning DNA**

Medical importance :

1- Drug resistance: R plasmid (resistance to (antibiotics) 2- Virulence: Enterotoxin production 3- Antimicrobial agents: as col plasmid Production of bacteriocin 4- Sex –factor: F plasmid (fertility factor)

A third source of genetic information in a bacterial cell can be provided by the presence of certain type of bacterial viruses:

BACTERIO PHAGES

1-ARE VIRUSES THAT PARASITIZE BACTERIA AND CONSIST OF NUCLIEC ACID CORE AND PROTIEN COAT

2-APHAGE PARTICLE MAY HAVE AT ITS CORE BESIDE ITS OWN NUCLIEC ACID AND ASEGMNT OF THE HOST DNA

Viral genome ss single stranded DNA or RNA may be linear or circular

- **They are unable to multiply in the absence of their bacterial host.**
- **Lethal to the host cell.**

Phage "life" cycles

Virulent/ Lytic cycle

➢ After a large number of progeny are built up inside the host, the bacterial host ruptures and phages are released.

Phage "life" cycles

Temperate/ Lysogenic cycle ➢ Occur integration of bacteriophage nucleic acid into host bacterium's genome.

➢ The Prophage " genetic material of bacteriophage" is transmitted to daughter cells at each subsequent division.

➢ Then a later events as (UV radiation or presence of chemicals) can release them, with proliferation of new phages via lytic cycle.

➢ Controlled replication without causing lysis called lysogeny .

➢ In this case bacteriophages becomes temporary part of the total genetic information .

Functions of Bacteriophages

1- Gene transfer: Transfer of genetic information from one bacterial strain to another by transduction.

2- Lysogenic conversion: In which phage s own DNA confers new properties to the cell. (Toxignicity of Coryn. Diphtheriae).

3- Bacterial typing : Phage typing is one of the methods used to identify bacteria

4- Genetic engineering.

Variations in Bacteriae

1-Phenotypic Variations

2-Genotypic Variations caused by two ways:

1-Gene transfer 2-Mutation

1-gene transfer via this processes :-

A- Transformation

b-Transduction

d-Conjugation

2-Mutation leads to two Variations :

1-Phenotypic Variations .

2-Non Phenotypic Variations.

Phenotypic variation Any change under the influence of environment ✓ Reversible ✓ Unstable

✓ Non heritable

Such as : loss of flagella

loss of cell wall

change of colony appearance

(smooth, rough)

Genotypic variation ✓ Irreversible ✓ Stable ✓ Heritable due to changes in nucleotide sequences

mutation

 The bacterial genome is normally identical in all the progeny Due to asexual binary fission (bacterial reproduction). Therefore DNA replication is very accurate
 But rarely inaccuracies produce a slight altered nucleotide sequence in one of the progeny cell Called mutation (Stable “Irreversible” and Heritable ” Passed to subsequent generations”)

Mutation can occur in 2 ways :

A-Spontaneously Rare 1 - 10^6 Change of DNA .

B-Induced by using certain chemical or physical agents (mutagens):

1- U.V rays 2- X- rays

3- H_2O_2 .

TYPES OF MUTATION

1-Multisite mutation 2- point mutation

1-Multisite mutation:

affecting one or very few Extensive chromosomal rearrangements

Inversion one to another

one or more Duplication

2- point mutation :

-Extensive - substitution - deletion

Types of mutation Mutation involving extensive chromosomal rearrangements such as :

Normal (wild type) A b c d e f g
1- inversion a b f d e c g
2- duplication a b c b c d
3- deletion a b e f
4- substitution a b x d e f g
5- insertion a b x c d e f g

Variation in Bacteria

II- Gene transfer

INCLUDES :

1-Transformation

2-Transduction

3-Conjugation

Transformation

Def.:

➢ bacterial cell can take small naked DNA fragments from its surroundings which brings about a genetic change in the recipient cell.

➢ Certain bacteria as(Bacillus, Haemophilus, Neisseria, Pneumococcus) can take up DNA from the environment & can be incorporated into the recipient 'chromosome.

➢ SIGNIFICANCE: INCREASE VIABILITY ,VIRULENCE,

Factors affecting transformation

DNA size state and Competence of the recipient cells

- some bacteria are able to take up DNA naturally only at particular time in their growth cycle when produce ds DNA of at least 5x 10^5 daltons

- Thus, transformation is sensitive to nucleases

Steps in transformation includes:

1- Uptake of DNA

Gram –ve bacteria is taken as dsDNA

Gram +ve bacteria Is taken as ss DNA The complementary is made in the recipient

2 – Gene recombination

After the donor DNA is taken up ,

a reciprocal recombination occurs between the chromosome & the donor DNA

- Recombination requires homology between the DNA& the bacterial recombination genes this called general recombination

transduction

Def.: Transfer of portion of DNA from one bacterial cell to another by bacteriophages .

➢ Bacterial viruses are involved in gene transfer by 3 mechanisms:

1-Generalized 2-abortive

3-specialized

Generalized transduction

➢ Generalized transduction is characterized by the transfer of a generalized group of genes
any gene on the bacterial chromosome have the same chance of being transferred by the viral carrier to another bacterial cell

➢ usually follows a lytic attack of a bacteriophage on bacterial cell, bacterial genome undergoes fragmentation often to a virus sized fragments of DNA

 which can be incorporated inside viral capsids (pseudovirus) can attack a new cell.

 - can be mediated by virulent phages or by temeprate phages

Abortive transduction

➢ In many generalized transduction , the segment of DNA from the donor cell fails to be integrated with the recipient chromosome.

➢ The bacterial DNA remains in the cytoplasm , it does not replicate but transmitted to one daughter cell following each division

Specialized transduction

➢ By which a specific set of genes is transferred by temperate phages from one bacterial cell into another

➢ Temperate phages are those capable of integrating their genetic material into the bacterial chromosome (lysogony)

➢ These phages recombine with the bacterial DNA at precise on the bacterial chromosome

The defective phage :

➢ It misses the viral genes where it left on the bacterial chromosome

➢ Can not replicate itself because of its missing genes

➢ Non defective phage must be present in the same cell (mixed infection) complement the missing segments of the defective virus so that it can be replicated & becomes a transducing phage.

Terms used

1 – Prophage : the viral DNA that integrates into the bacterial chromosome

2 – Temperate phage : Viruses that either lyse the cell or behave as a prophage Can reproduce by 2 mechanisms (the lytic cycle & the lysogenic cycle).

3 – Virulent phage : viruses that only lyse the cell

4- Transducing phage: It is always defective not able to lysis of the infected cell Capable of mobilizing bacterial genetic material from one cell to another .

Plasmid-mediated conjugation (conjugation)

Def of conjugation :

- Is the transfer of DNA from a donor to a recipient by direct physical contact between the cells
- Donor (male) & recipient (female)
- The direction of transfer is one way (donor to recipient)

Mechanism of bacterial conjugation

1- donor cell produce pilus
2 – pilus attaches to recipient cell , brings the 2 cells together
3- the mobile plasmid is nicked & a single strand of DNA is then transferred to the recipient cell
4- both cells recircularize their plasmids, synthesize second strands & reproduce pili, both cells are now viable donors

Recombination

- Plasmid DNA can be integrated into bacterial chromosome It is known as episome
- N.B.:

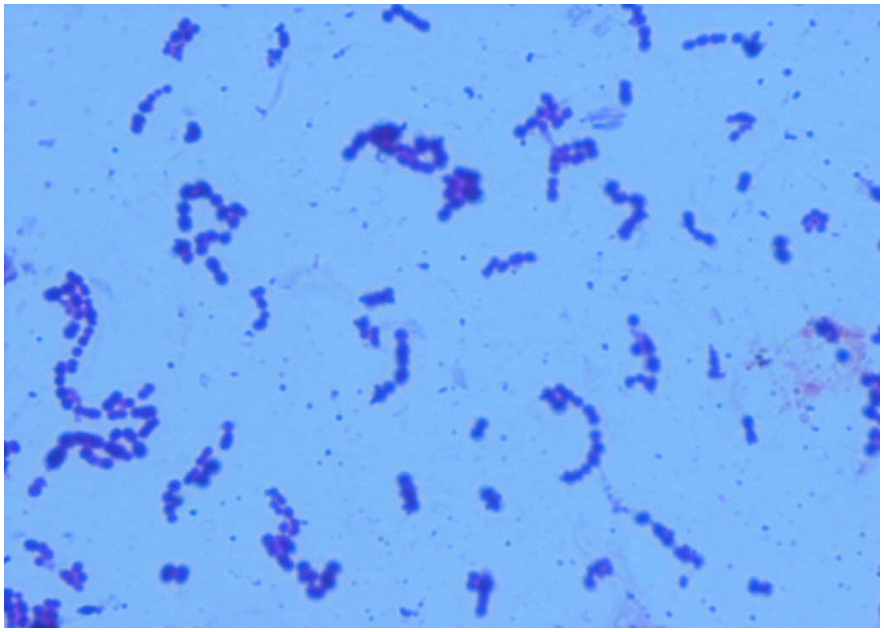
➢ Plasmids of gram positive are non transmissible plasmids as they lack self transfer because they lack genes that code for the sex pili.

Transposons (Jumping genes)

- These are genetic materials that can move (Jump or transposon) from one location to another on the same chromosome or from plasmid to plasmid or from plasmid to chromosome and the reverse
- They can not autonomously replicate but they replicate with the plasmid or chromosome
- Transmissible , Insertion into plasmid can lead to its dissemination

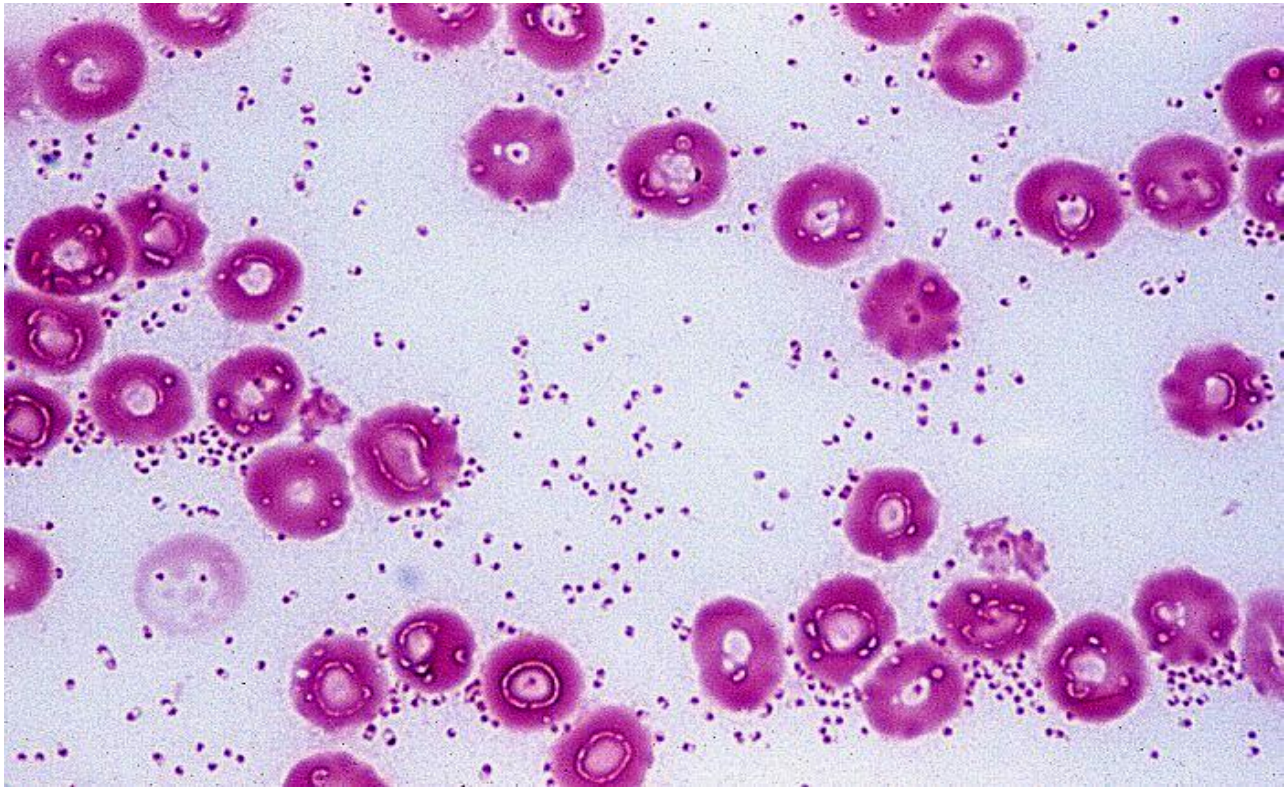
JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
28109091800516 - 2004578801966

Blood smear from an animal that has died of anthrax. Stained with polychrome methylene blue, showing purple capsule and blue bacilli (MacFadyean stain).

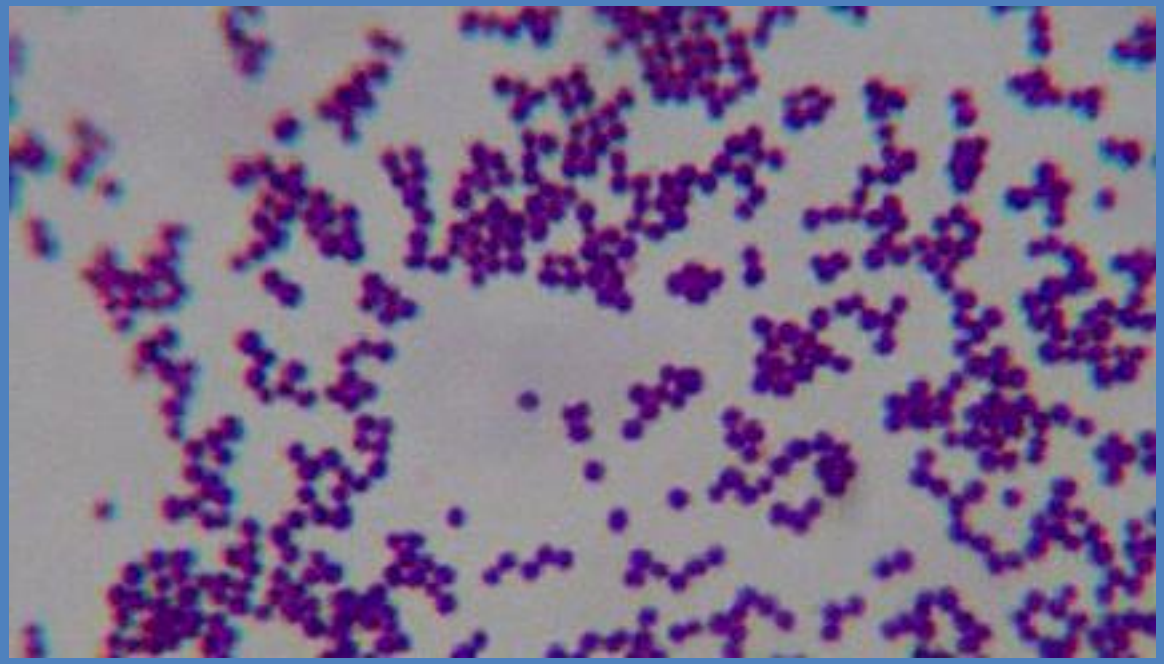


Streptococcus agalactia stained with loffler's methylene blue, showing blue cocci arranged in short chain

Streptococcus pyogen **stained with loffler's methylene blue, showing blue cocci arranged in long chain**

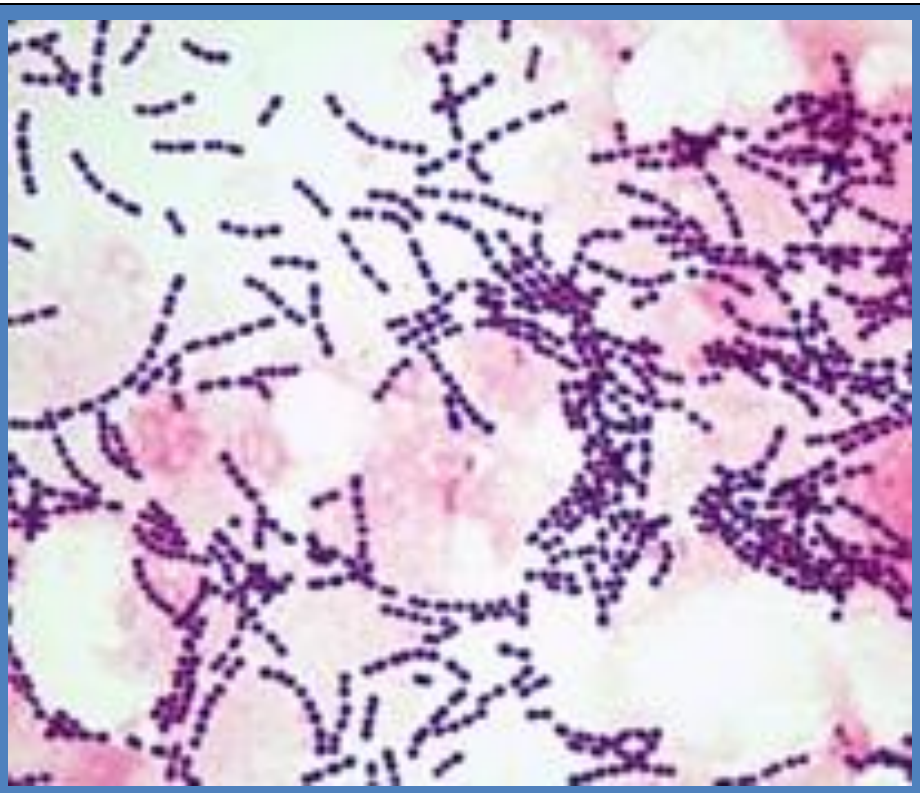


Blood film stained with leishman's stain showing bipolarity of *pasteurella multocida*



Staphylococci stained with gram's stain showing

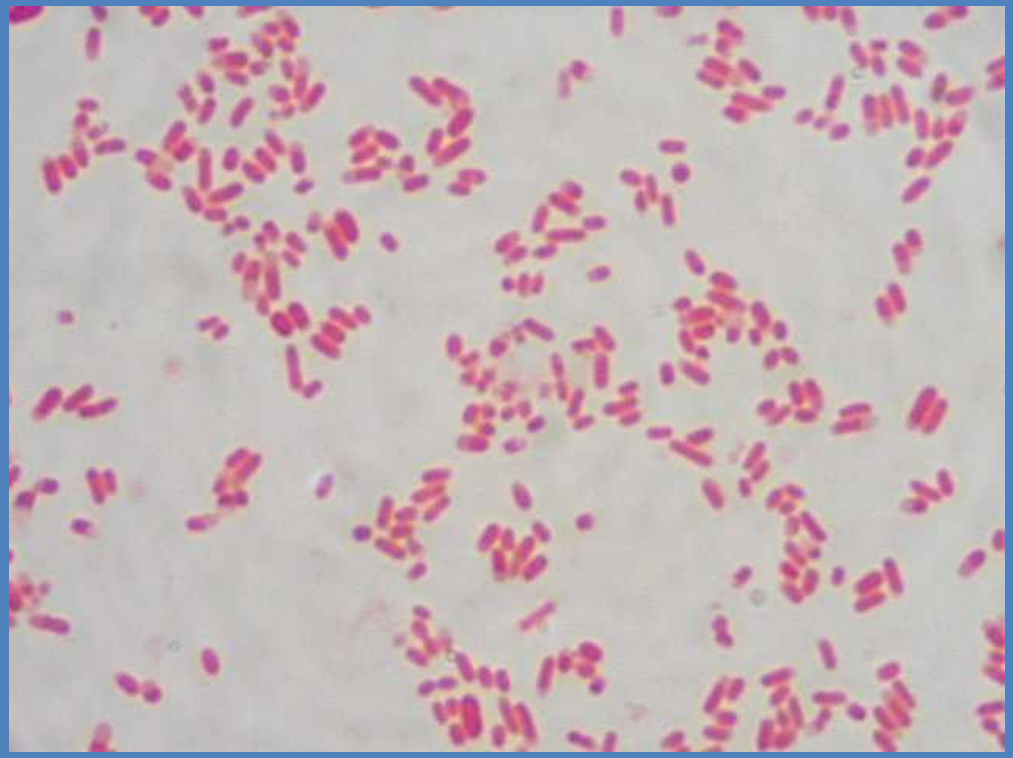
- **Gram positive cocci arranged in grape like clusters**



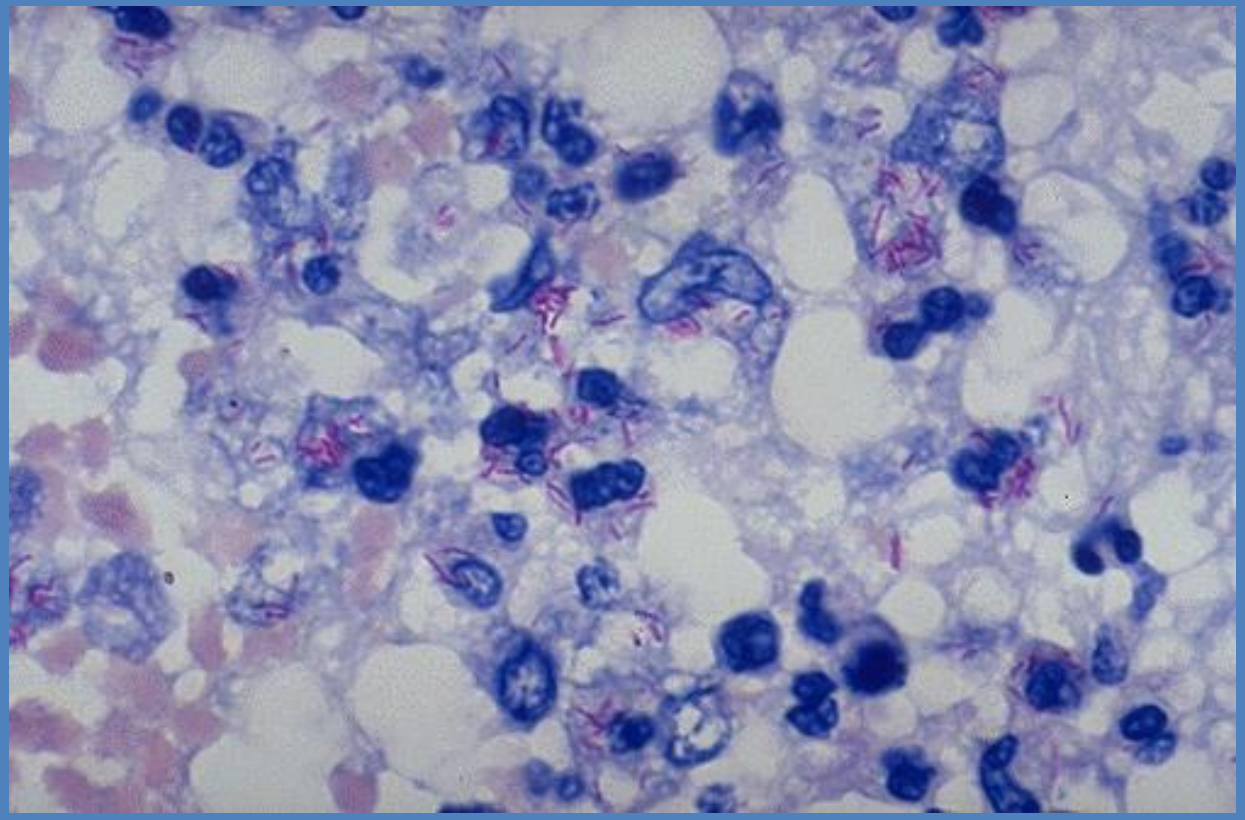
Streptococci stained with gram's stain showing gram positive cocci arranged in chain

Clostridium tetani stained with gram's stain showing

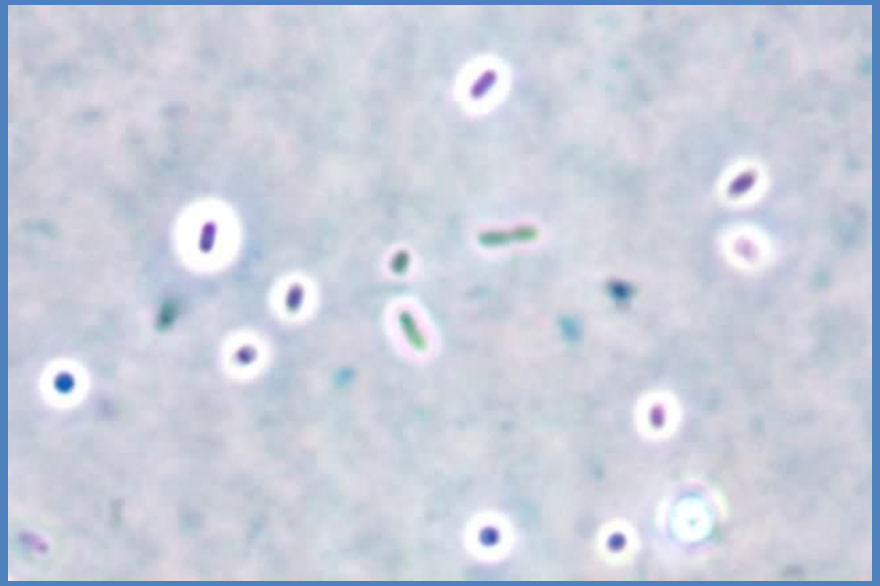
gram positive bacilli with drum stick appearance due to terminal endo-spore



Gram's stain showing gram negative cocco-bacilli (red color) arranged in clusters



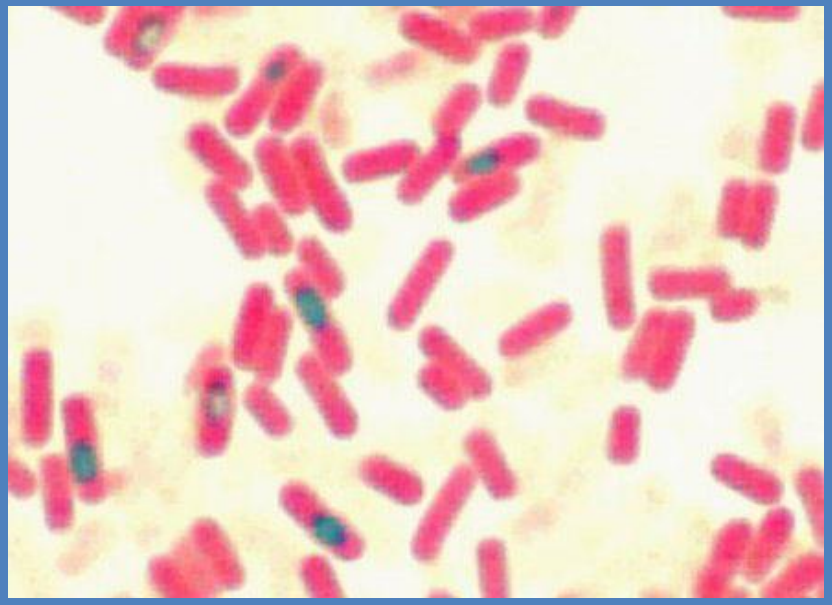
Mycobacterium tuberculosis **stained with ziehl neelsen stain showing red long thin bacilli arranged in clusters (acid fast bacilli) against blue background.**



Dry India ink preparation showing capsulated bacteria (the capsule appear as colorless zone around the blue bacterial body and against blue back ground)

Bacillus anthracoids **stained with gram stain showing**

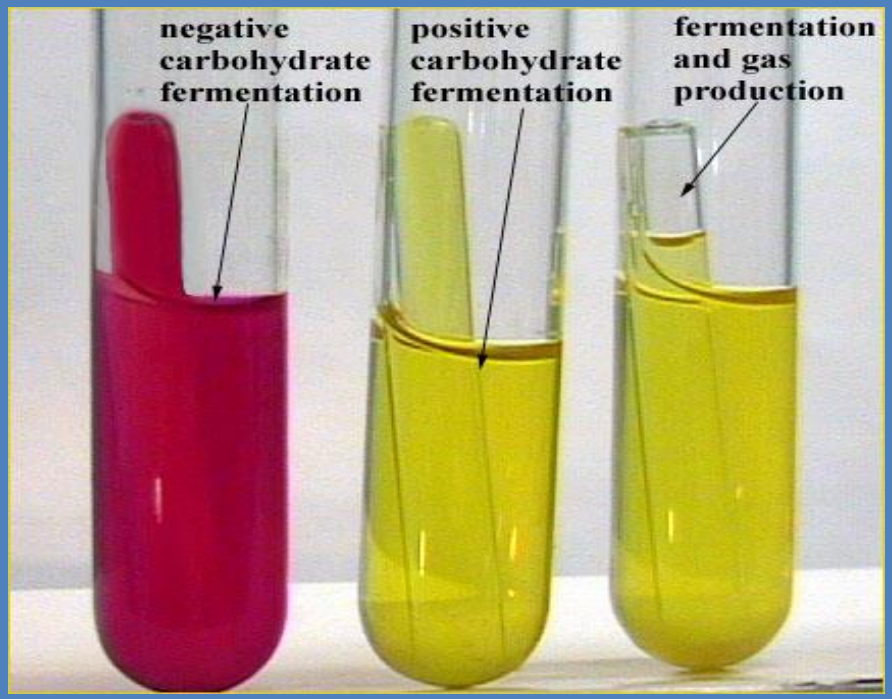
gram positive bacilli arranged in long chain and with central unstained spore.



Bacillus anthracis **stained with malachite green stain showing**

red bacilli with green central spores

arranged in single or pair



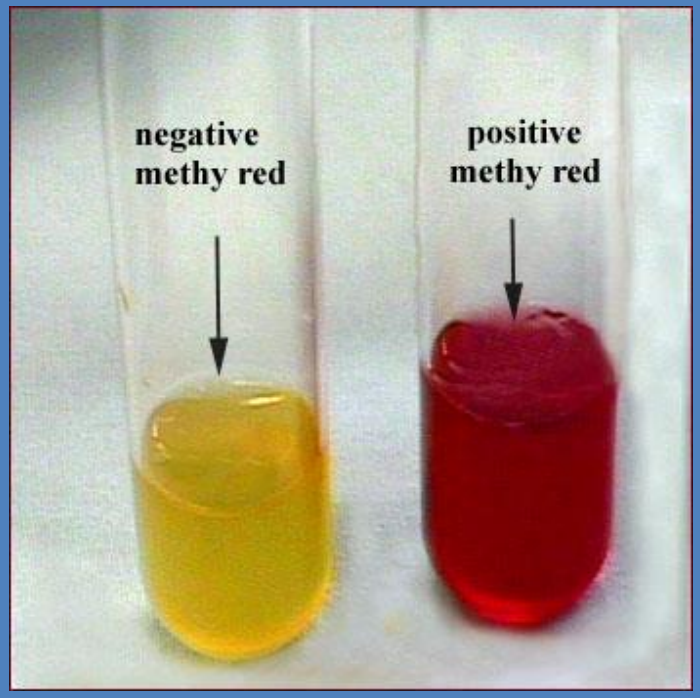
Sugar fermentation test showing, from left to right

- Red color indicate negative sugar fermentation test
- Yellow color without presence of gases in durham's tube indicate positive result (fermentation without gas production)
- Yellow color with presence of gases in Durham's tube indicate positive result (fermentation with gas production)

Indole production test showing from left to right

red ring: positive result

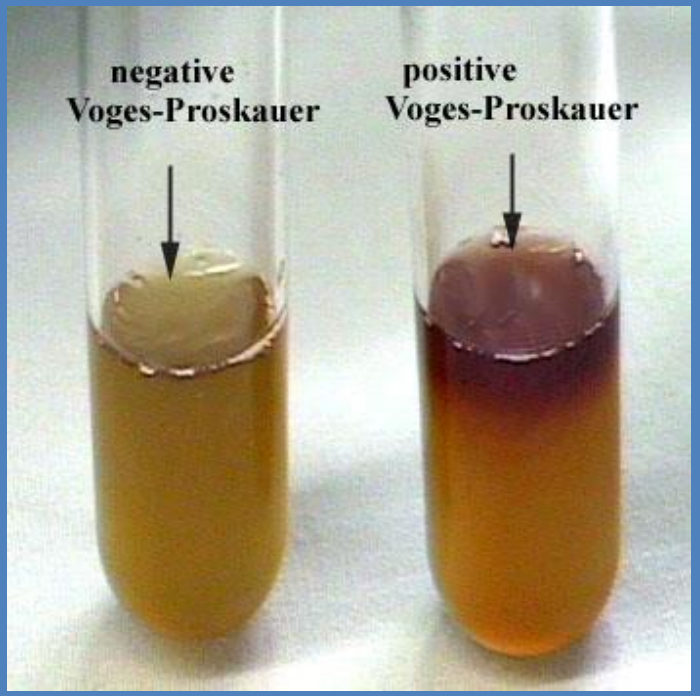
no red ring: negative result



Methyl red test showing from left to right

Yellow color indicate negative result

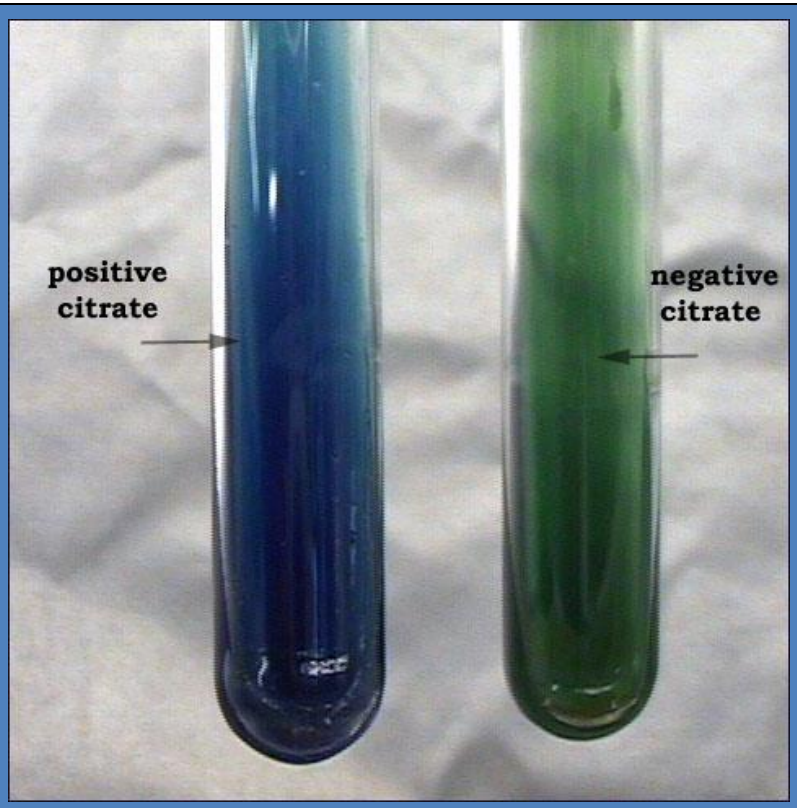
Red color indicate positive result



Voges proskauer test showing from left to right

Yellow to copper color indicate negative result

Dark red color indicate positive result



Citrate utilization test showing from left to right

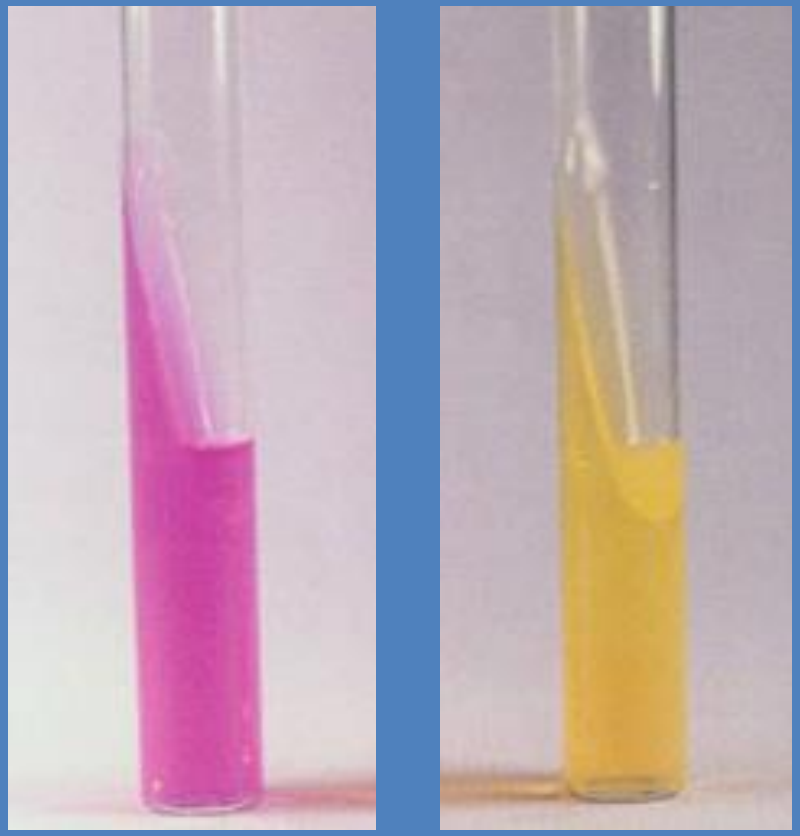
blue color indicate positive result

green color indicate negative result

H_2S production test showing from left to right

No black color indicate negative result

Black color indicate positive result



<u>Urea hydrolysis test showing from left to right</u>
pink (foshia) color indicate positive result
Yellow color indicate negative result

Sugar fermentation: +ve **indole : +ve** **MR: +ve**

VP:-ve **citrate : -ve** **urea: -ve**

H_2S: -ve

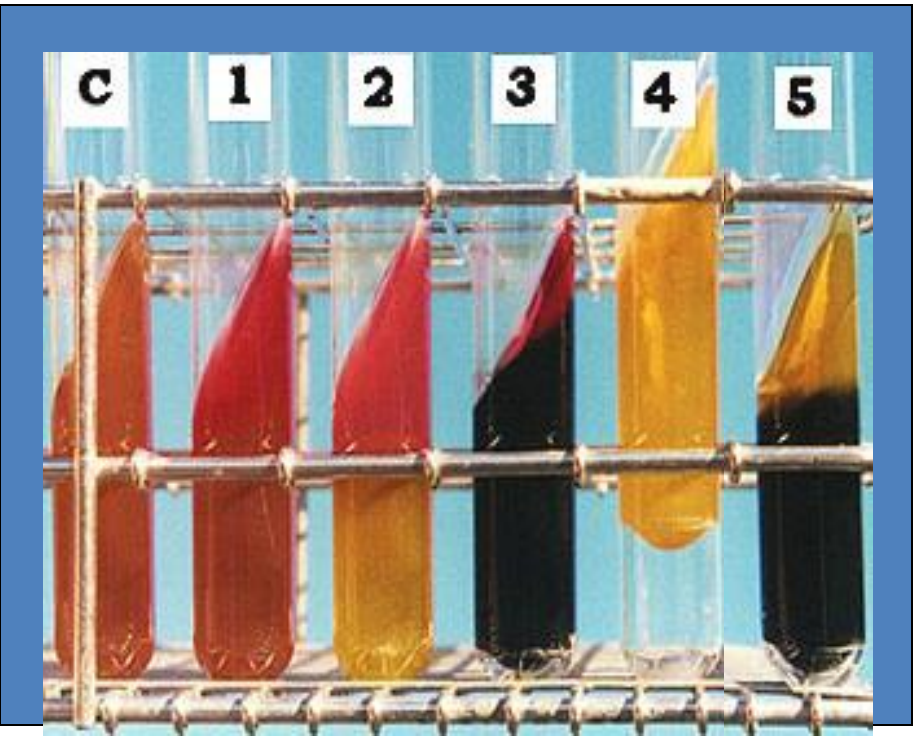
The tested microrganism is *E.coli*

Sugar fermentation: -ve **indole : +ve** **MR: +ve**

VP:-ve **citrate : -ve** **urea: -ve**

H_2S: -ve

The tested microrganism is *shigella*



Triple Sugar Iron (TSI) Agar showing from left to right →

C : Uninoculated control (orange color)

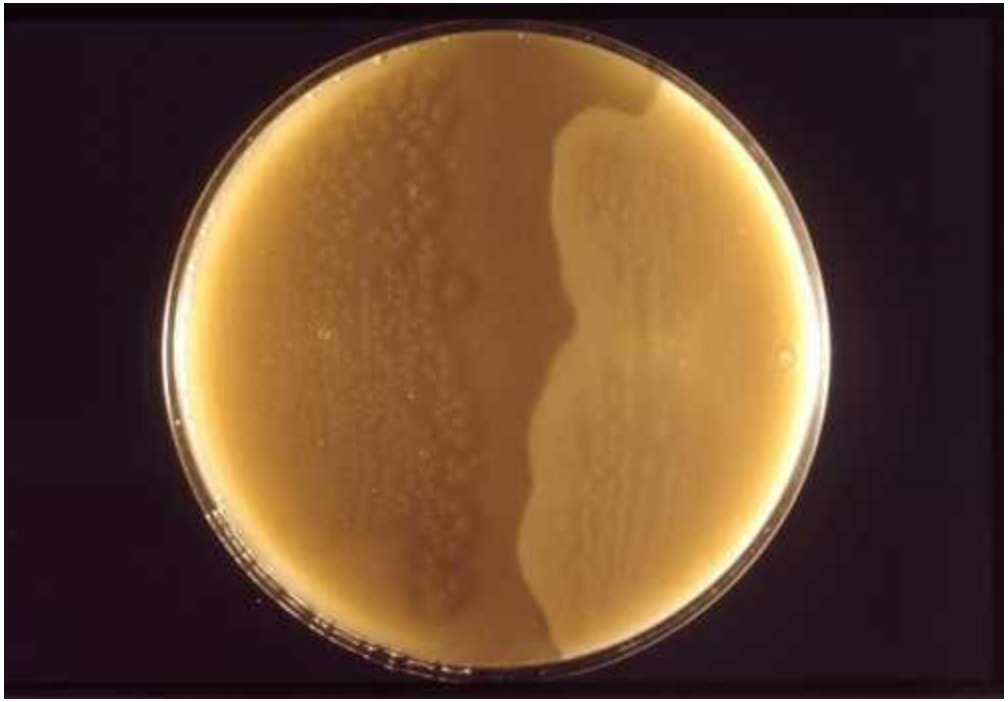
1: the color change to red color due to de-amination of amino acid and production of alkaline end product. As *Pseudomonas*

2: the pottom of the slant change to yellow color due to fermentation of glucose and production of minor amount of acids. As Shigella

3: black color due to H_2s production. As *Salmonella, Edwardsiella*

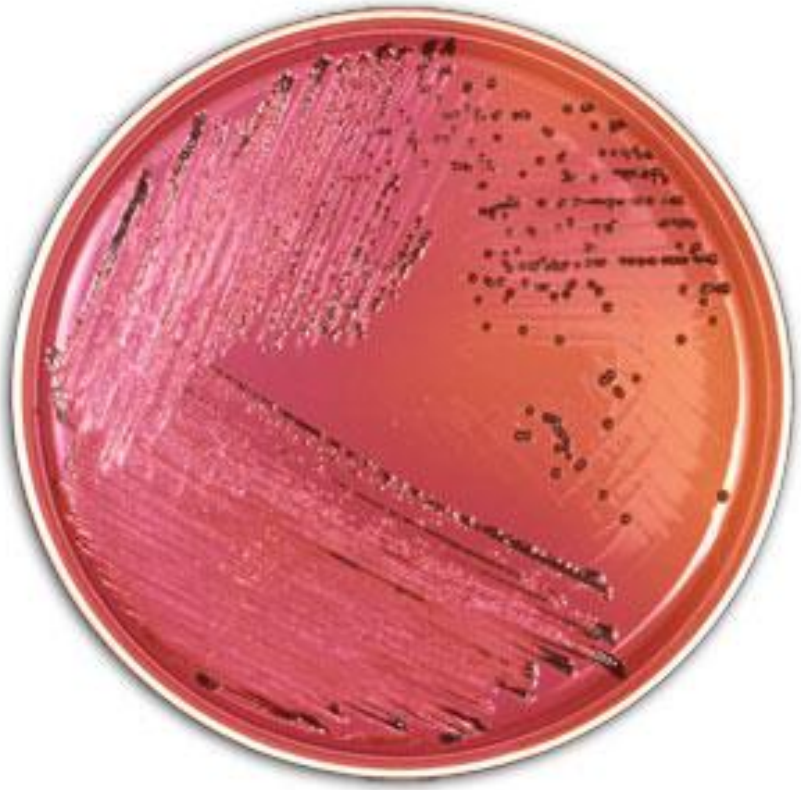
4: Yellow slant, yellow butt and lifting and/or cracking of media, no black coloration due to Lactose, sucrose and glucose fermented, H2S has not been produced but gas has been produced, as *E. coli, Enterobacter, Klebsiella*

5: Yellow slant, yellow butt and no lifting nor cracking of media, black coloration due to Lactose, sucrose and glucose fermented without gas production, H2S production, as *Citrobacter*

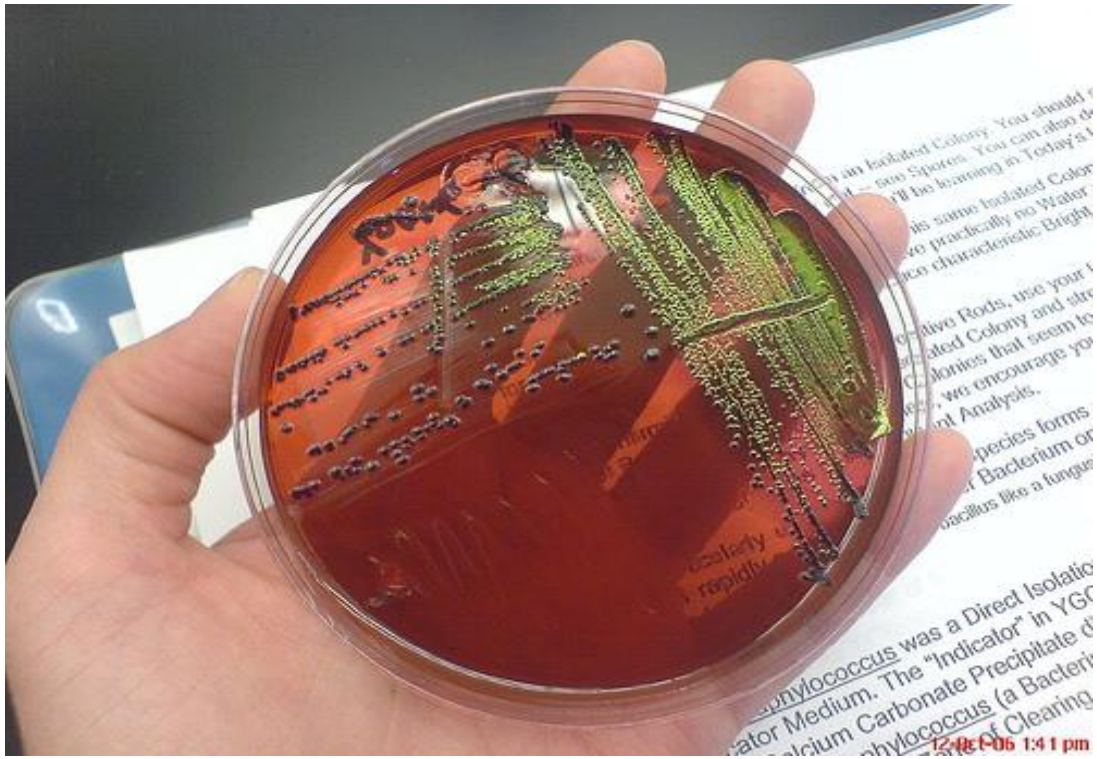


Egg yolk media cultivated with Cl. Welchii showing opalescence due to lecithinase activity

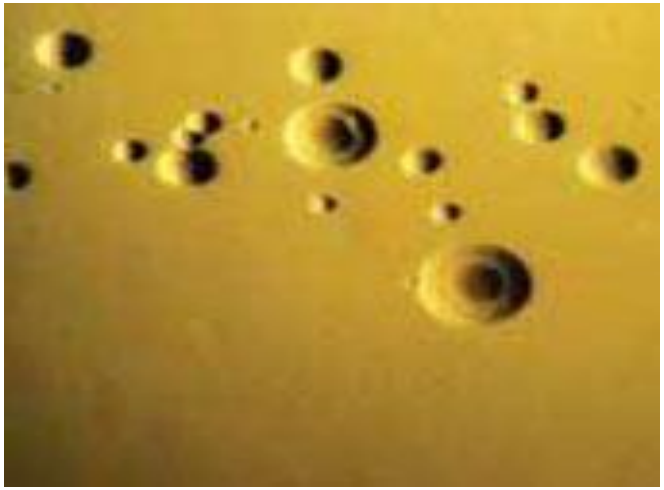
(positive Nagler reaction)



- XLD media cultivated with Salmonella showing red colony with black center



EMB agar (eosin methylene blue) cultivated with E.coli showing metalic green colony)



Microscopic colonies of mycoplasma which appeared as frayed egg appearance

Elke's test (-ve)

Elek test for corynebacterium diphtheria-Toxigenic strain

مرض الكبد الدهنى غير الكحولى

التشخيص :

لأن مرض الكبد الدهني غير الكحولي لا يُسبِّب أي أعراض في معظم الحالات، فإنه كثيرًا ما يتمُّ الانتباه إليه طبيًّا عندما تُشير اختبارات تُجرَى لأسباب أخرى إلى مشكلة في الكبد. ويُمكن أن يحدث ذلك إذا كانت حالة الكبد تبدو غير طبيعية عند الفحص بالموجات فوق الصوتية أو إذا كانت نتائج اختبار إنزيم الكبد لديكَ غير طبيعية.

وتشمل الاختبارات التي تُجرَى لمعرفة التشخيص وتحديد مدى شدة المرض ما يلي:

إختبارات الدم

الإجراءات التصويرية

فحص نسيج الكبد

إختبارات الدم:-

تعداد الدم الكامل
اختبارات إنزيمات الكبد ووظائف الكبد
اختبارات التهاب الكبد الفيروسي المزمن (التهاب الكبد A، التهاب الكبد C وغيرهما)
اختبار فحص الداء البطني
سكر الدم الصائم
الهيموغلوبين السكري A1C، والذي يوضح مدى ثبات سكر الدم لديك
فحص الدهون، والذي يقيس دهون الدم، مثل الكوليسترول والدهون الثلاثية

الإجراءات التصويرية:-

تشمل الإجراءات التصويرية المُستخدمة في تشخيص مرض الكبد الدهني غير الكحولي ما يلي:

- تصوير البطن بالموجات فوق الصوتية (الألتراساوند)،

- وهو الاختبار الأول الذي يُجرى عادةً في حالة الاشتباه في الإصابة بأمراض الكبد.

•فحص التصوير المقطعي المُحوسب أو التصوير بالرنين المغناطيسي للبطن.

• تفتقر هذه التقنيات القدرة على تمييز التهاب الكبد الدهني الكحولي من مرض الكبد الدهني غير الكحولي، ومع ذلك قد تظل تُستخدم.

•تصوير المرونة العابر، وهو نمط مُعزز من الموجات فوق الصوتية التي تقيس درجة تيبُّس الكبد لديك. يُشير تيبُّس الكبد لديك إلى وجود تليُّف أو تندُّب.

•تصوير المرونة بالرنين المغناطيسي، يعمل من خلال الدمج بين التصوير بالرنين المغناطيسي والموجات الصوتية لتكوين خريطة مرئية (مُخطط المرونة) تُظهر درجة تيبُّس أنسجة الجسم

فحص نسيج الكبد:-

إذا كانت الفحوصات الأخرى غير قاطعة، فقد يُوصي طبيبك بإجراء لأخذ عينة نسيج من الكبد (خزعة الكبد). تُفحص عينة النسيج في المختبر بحثًا عن علامات الالتهاب والتندُّب.

قد يكون إجراء خزعة الكبد غير مريح، وينطوي على خطورة منخفضة سيُراجعها الطبيب معك بالتفصيل. يُنفذ هذا الإجراء عن طريق إدخال إبرة عبر جدار البطن إلى الكبد.

العلاج:-

بداية العلاج عادةً هي فقدان الوزن من خلال الجمع بين اتِّباع نظام غذائي صحيٍّ، وممارسة التمارين الرياضية. يعالج فقدان الوزن الأسباب التي تُسهِم في الإصابة بمرض الكبد الدهني غير الكحولي. ومن الناحية المثالية، يكون فقدان ١٠% من وزن الجسم أمرًا مرغوبًا فيه، ولكن التحسن في عوامل الخطر يمكن أن يتضح إذا فقدْتَ من ٣ إلى ٥% من وزنك الأولي. وتمثل جراحة إنقاص الوزن أحد الخيارات أيضًا لأولئك الذين يحتاجون لفقد الكثير من الوزن.

قد تكون زراعة الكبد خيارًا لأولئك الذين لديهم تشمع بالكبد؛ نتيجة الإصابة بالتهاب الكبد الدهني غير الكحولي.

العلاجات المحتمَلة في المستقبل :-

لايوجد أي علاج بعقاقير معتمدة من إدارة الغذاء والدواء الأمريكية لمرض الكبد الدهني غير الكحولي، ولكن أُجريت دراسات على بعض الأدوية وكانت ذات نتائج واعدة.

بمساعدة طبيبكَ، يُمكنكَ اتخاذ خطوات لإحكام السيطرة على مرض الكبد الدهني غير الكحولي. يُمكنك:

إنقاص الوزن. في حالِ كنتَ مصابًا بزيادة الوزن أو السمنة، فقُمْ بتقليل عدد السعرات الحرارية التي تتناولها كل يوم، وقُمْ بزيادة نشاطكَ البدني من أجل إنقاص وزنك. تخفيض السعرات الحرارية هو مفتاح إنقاص الوزن والتحكُّم في هذا المرض. إن حاولتَ إنقاص الوزن في السابق، ولكنكَ لم تنجحْ، فاطلبِ المساعدة من طبيبك.

اخترْ نظامًا غذائيًّا صحيًّا. اتبعْ نظامًا غذائيًّا صحيًّا غنيًّا بالفواكه والخضراوات والحبوب الكاملة، وراقِبْ جميع السعرات الحرارية التي تتناولها.

مارِسِ الرياضة وكن أكثر نشاطًا. حاولْ تخصيص ٣٠ دقيقة على الأقل لممارسة التمرينات الرياضية معظم أيام الأسبوع.

إذا كنتَ بصدد محاولة إنقاص وزنكَ، فستجد أن قيامكَ بالمزيد من التمارين أمر مفيد ونافع. ولكن إذا لم تمارسْ فعلًا التمارين بانتظام، فاحصُلْ على موافقة طبيبكَ أولًا وابدأْ ببطء.

سيطِرْ على مرض السكري. اتَّبِعْ تعليمات طبيبكَ لتبقى مسيطرًا على مرض السكري الخاص بك. تناوَلِ الأدوية الموصوفة لكَ حسب الإرشادات وراقِبْ عن قرب سكر الدم لديك؟

قُمْ بتقليل الكوليسترول. إن اتباع نظام غذائي صحي يعتمد على النباتات وممارسة الرياضة، وتناول الأدوية يُمكن أن يُساعد في الحفاظ على الكوليسترول والدهون الثلاثية عند مستويات صحية.

حماية كبدك. تجنَّبِ الأشياء التي من شأنها أن تضع مزيدًا من الضغوط على الكبد. فعلى سبيل المثال، لا تتناوَل المشروبات الكحولية. اتَّبِعِ التعليمات الخاصة بالأدوية الموصوفةَ لكَ وأيضًا الأدوية المتاحة دون وصفة طبية. تشاوَرْ مع طبيبكَ قبل استخدام أي علاجات عشبية؛ لأنه ليس كل المنتجات العشبية آمنة.

Clinical Pathology

Section WRITTEN AND PRESENTED BY

Doctor

Mohammed Mahmoud

Mohammed Yousef Wehaba

Flow cytometry

Introduction:

Flow cytometry (FCM) is a powerful, rapid and cost effective analytic technique in which cell suspension is stained with fluorescent labeled antibodies and subjected to analysis on a specialized instrument called flow cytometer.

Uses: Flow cytometry is useful for identifying and counting single cells labeled by fluorochrome monoclonal antibodies to specific surface or intracellular antigens. It provides detailed multiple intrinsic parameters of cells of normal, reactive, benign and neoplastic tissue. Thus, it can detect the:

- Cell lineage (e.g. B lymphocyte versus T lymphocyte).
- Degree of maturation (e.g. pre-B or mature B cells).
- Cell function (presence of cytokines or other receptors).

Specimen

- Fresh, unfixed tissue specimen from which single cell suspension can be generated is suitable for flow cytometric analysis.
- The cell suspension may be peripheral blood, bone marrow aspirates and core biopsies, fine needle aspirates, fresh tissue biopsies (e.g., lymph node) and body fluids.

Components of Flow cytometry

It is a relatively complex device with four major components.

- Fluidics system

1. Specimen stream: Specimen is subjected to form a flowing stream of fluid such that the suspended cells move as single cell suspension in the central column.
2. Sheath fluid: Specimen stream is surrounded by buffered saline known as sheath fluid, which maintains the monolayer of cells in the center of the flowing stream. This stream of single cells pass through the laser light.

- Optics system (Optics and light detectors)

1. Light source:
   - The light source (usually lasers) illuminates the fluorochrome tagged cells, which are propelled through the flow chamber.
   - The light hitting the cells is either scattered or absorbed and then re-emitted (fluorescence).
   - The pattern of scattered or absorbed laser light is dependent on cell size, shape, volume and granularity. Thus, it gives information about the intrinsic cellular parameters.
2. Detectors:

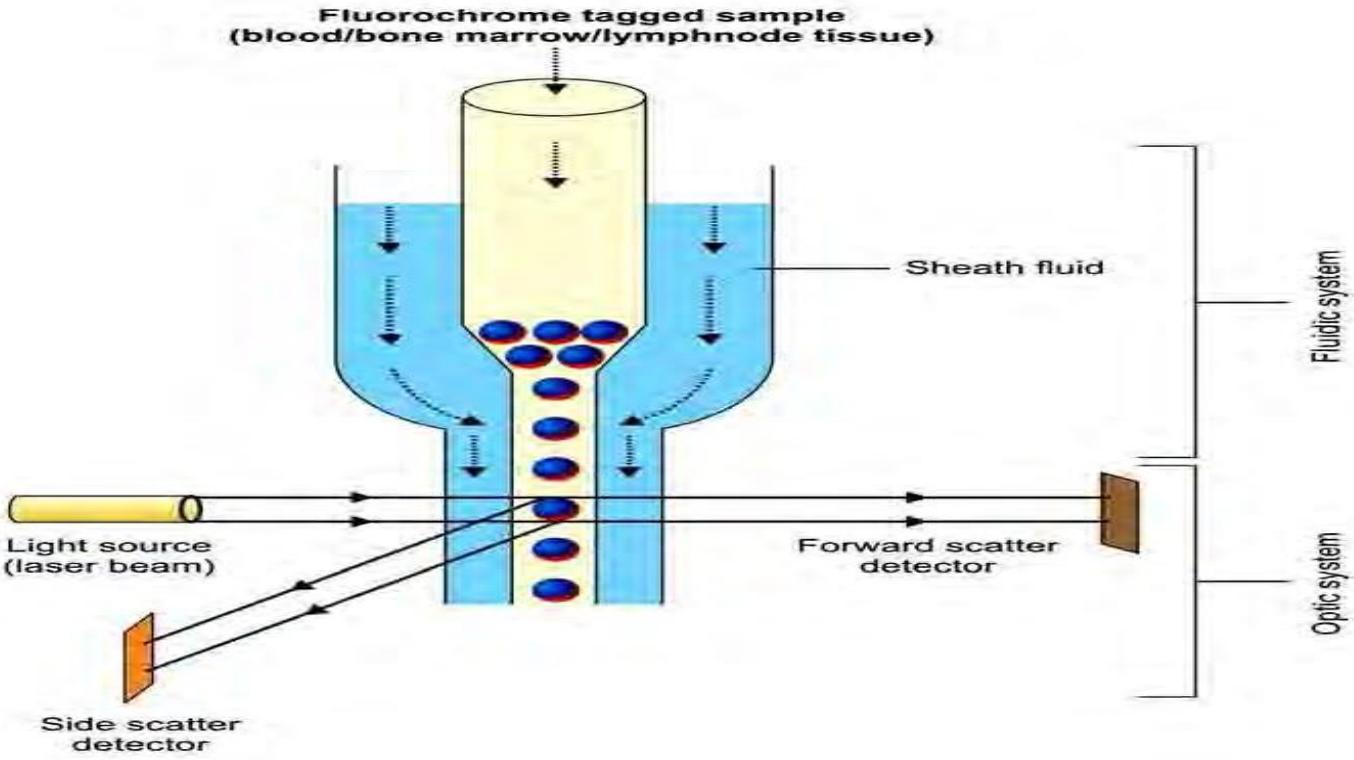
- The light scattered or emitted by the cells as cells flow through the laser beam is measured by detectors. In addition, fluorescent molecules or fluorochromes attached to the cells emit fluorescence.
- Detectors convert the detected light into electrical impulses, the magnitude of which are proportional to the amount of light and gives information about the cross-sectional area of the cell.

- Electronic system

  Measures electrical impulses and converts it into digital information that is gathered and interpreted by the analysis software. These profiles of cells are normally displayed as dot plots or histograms.

- Computer

  Which controls the functions of flow cytometer.



Applications of flow cytometry

Flow cytometry is used in a variety of different fields including immunology, pathology and medicine. Common applications are:

1. Erythrocyte Applications
   a. Reticulocyte count and maturation index (Thiazole orange enters erythrocytes, binds to RNA, and fluoresces when exposed to laser light).

b. Erythrocyte-bound immunoglobulin (The technique involves incubation of washed erythrocytes with a species-specific fluorescein-labeled anti-IgG or anti-IgM).

c. Detection of erythrocytic parasites (e.g. plasmodium, trypanosomes, Babesia, Haemobartonella, Eperythrozoan, and Anaplasma).

2. Platelet Applications

a. Reticulated platelets (reticulated (Immature) platelet numbers can be used to determine whether thrombocytopenia is caused by decreased production or consumption/destruction). Residual RNA in immature platelets can be quantified by incubating platelets with thiazole orange.

b. Anti-platelet antibodies: In the direct test, platelets are isolated from blood, washed to remove residual plasma, and incubated with a species-specific anti-IgG or anti-IgM. Positive results occur in suspected immune-mediated thrombocytopenia. Also it indicates ehrlichiosis and malignancy

c. Activated platelets: flow cytometric tests detect circulating activated platelets. These tests include binding of fibrinogen to platelets, binding of monoclonal antibodies to activation-dependent markers on the platelet surface,

detection of platelet microparticles, detection of shape change, and detection of platelet–leukocyte aggregates

3. Leukocyte Applications
   a. Immunophenotyping leukocytes: involves the use of specific antibodies and fluorochrome reagents to identify specific cell types in blood and bone marrow
   b. Immunophenotyping leukemias and lymphomas: Classification of hematopoietic neoplasms: Leukemia, lymphoma, myelodysplasia and systemic mastocytosis.
   c. Assessment of neutrophil function: These include tests for oxidative metabolism, phagocytosis, degranulation, activation markers, surface expression of secreted granule contents, shape change, F-actin content, and opsonization.
4. Bone Marrow Applications
   a. Differential cell counts: bone marrow differential cell counts were performed by labeling cells with the fluorochrome 27-dichlorofluorescin-diacetate. This technique was able to identify populations of proliferating and mature myeloid cells, proliferating and mature erythroid cells, and megakaryocytes.
   b. Maturation and proliferative activity: Mitotic activity causes slight variations in the DNA content of cells, which results in altered fluorescence. Mitotic activity is usually increased in acute leukemias

WORLD MAP
1. Netherlands
2. Belgium
3. Luxembourg
4. Switzerland
5. Slovenia
6. Croatia
7. Bosnia and Herzegovina
8. Czechia
9. Slovakia
10. Austria
11. Hungary
12. Serbia
13. Moldova
14. North Macedonia
15. Albania
16. Cyprus
17. Lebanon
18. Guinea-Bissau
19. Guinea
20. Ghana
21. Togo
22. Benin
23. Cameroon
24. Equatorial Guinea
25. Rwanda
26. Cambodia
27. Panama
28. Malawi
29. Liechtenstein
30. Montenegro
31. Kosovo
32. Palestinian Territories
33. St. Vincent & the Grenadines
0 2,000 4,000 Miles
0 2,000 4,000 6,000 8,000 Kilometers
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(SPONDYLOSIS)

(داء الفقار) (خشونة الفقرات)

مساعدباحث /محمدمحمودمحمديوسف وهيبه

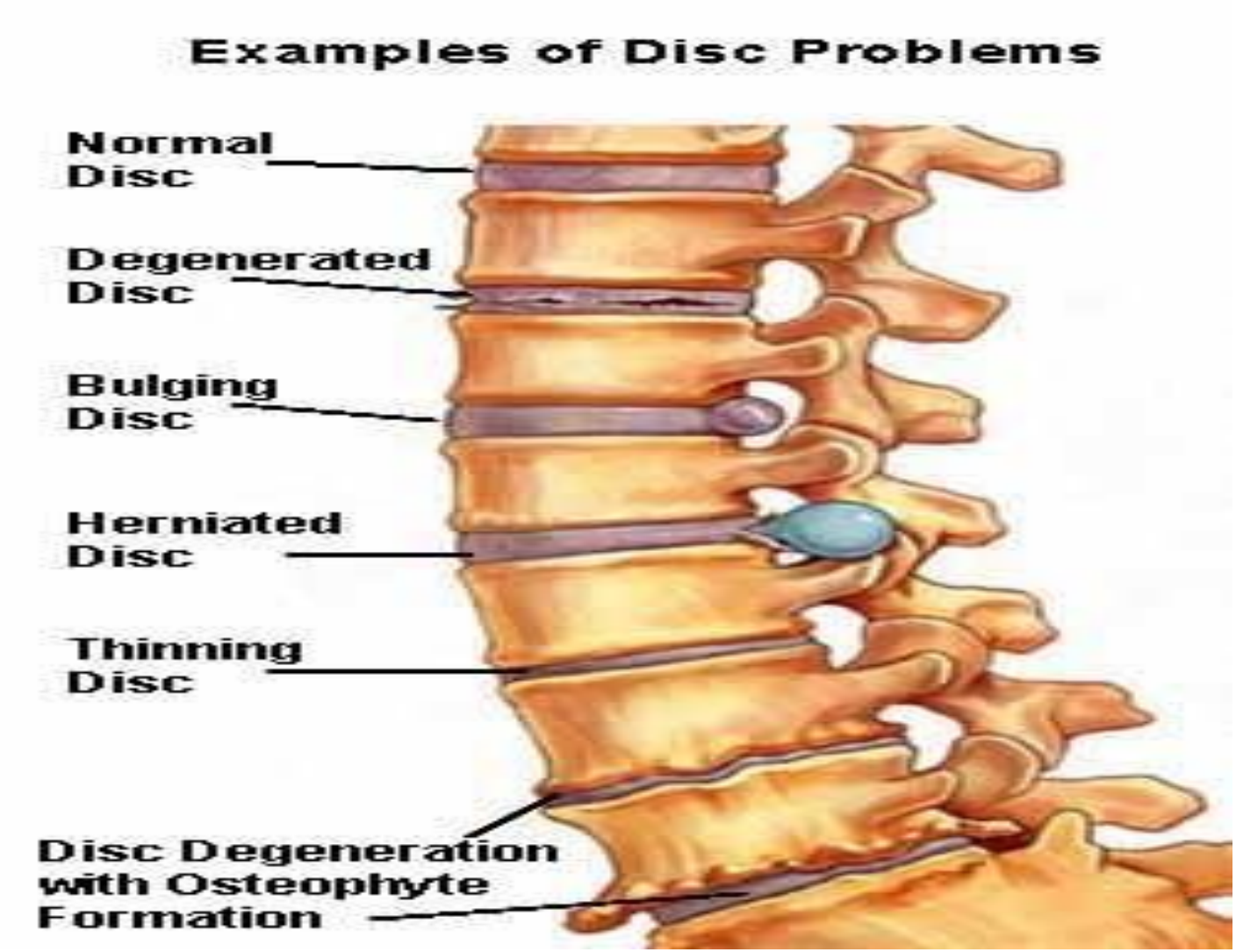
معهدبحوث الصحة الحيوانية

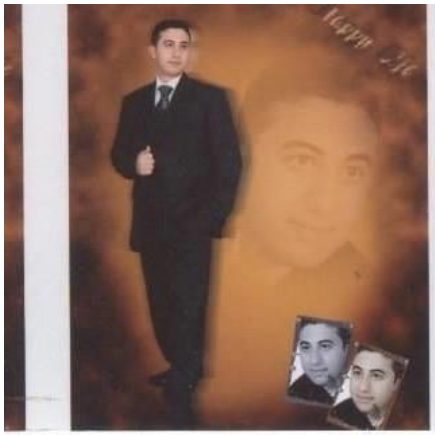
مركزالبحوث الزراعية

وزارة الزراعة

جمهورية مصرالعربية

يُعد داء الفقار الرقبية (خشونة الفقرات) مصطلحًا عامًا يصف التلف والتمزق المرتبط بالمرحلة العمرية والذي يصيب أقراص العمود الفقري في الرقبة.
وتظهر علامات الفُصال العظمي كلما أصاب الجفاف والانكماش الأقراص، بما يتضمن النتوءات العظمية على طول أطراف العظام. ويُعد داء الفقار الرقبية منتشرًا للغاية ويتفاقم مع التقدم في العمر.

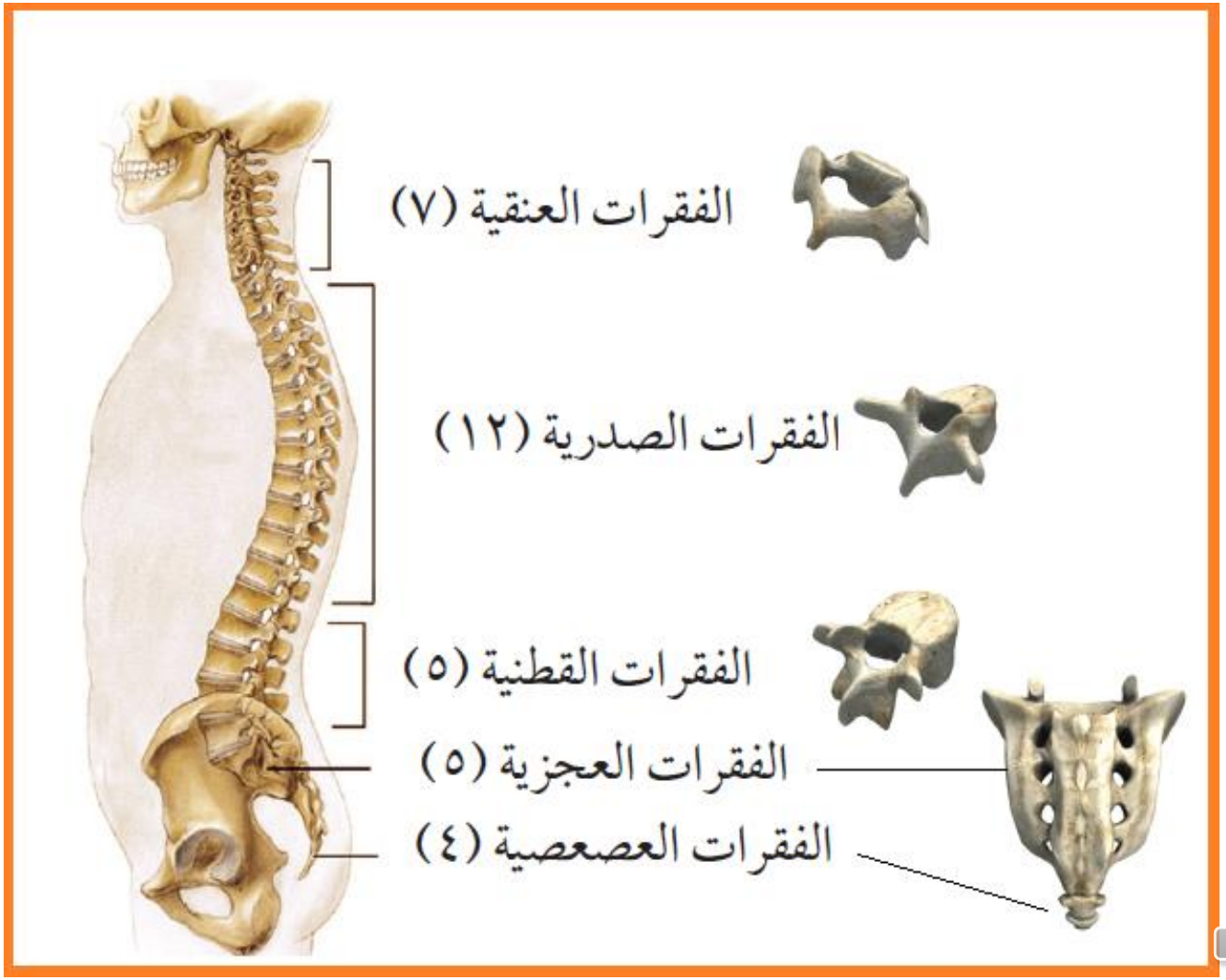

Examples of Disc Problems
Normal Disc
Degenerated Disc
Bulging Disc
Herniated Disc
Thinning Disc
Disc Degeneration with Osteophyte Formation



الفصال العظمى

ويُعرَف الالتهاب المفصلي العظمي في أغلب الحالات بالتآكل والاهتراء الناجمين عن استخدام المفصل بمرور الزمن. ولا يستهدف الالتهاب المفصلي العظمي النسيج الغضروفي وحسب، بل يؤثر كذلك على المفصل بالكامل. ويسبب تغيرات في العظام وتآكل الأنسجة الضامة التي تربط المفصل معًا وتربط العضلات بالعظام.

العمود الفِقْرِي أو العمود الفَقْرِي أو الفَقَار للكائنات الحية الفقارية مكون من سلسلة فِقْرَات مفرغة لحفظ النخاع الشوكي الذي يمتد فيها ويبلغ عدد الفِقْرَات عند الإنسان ٣٣ فقرة. يدعى الفراغ الذي يسكنه النخاع الشوكي بالنفق الفقري.


الفقرات العنقية (٧)
الفقرات الصدرية (١٢)
الفقرات القطنية (٥)
الفقرات العجزية (٥)
الفقرات العصعصية (٤)

لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة. اليكم لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة تشكل القسم السفلي من العمود الفقري (العجز والعصعص).

يقسم العمود الفقري إلى المناطق التالية:

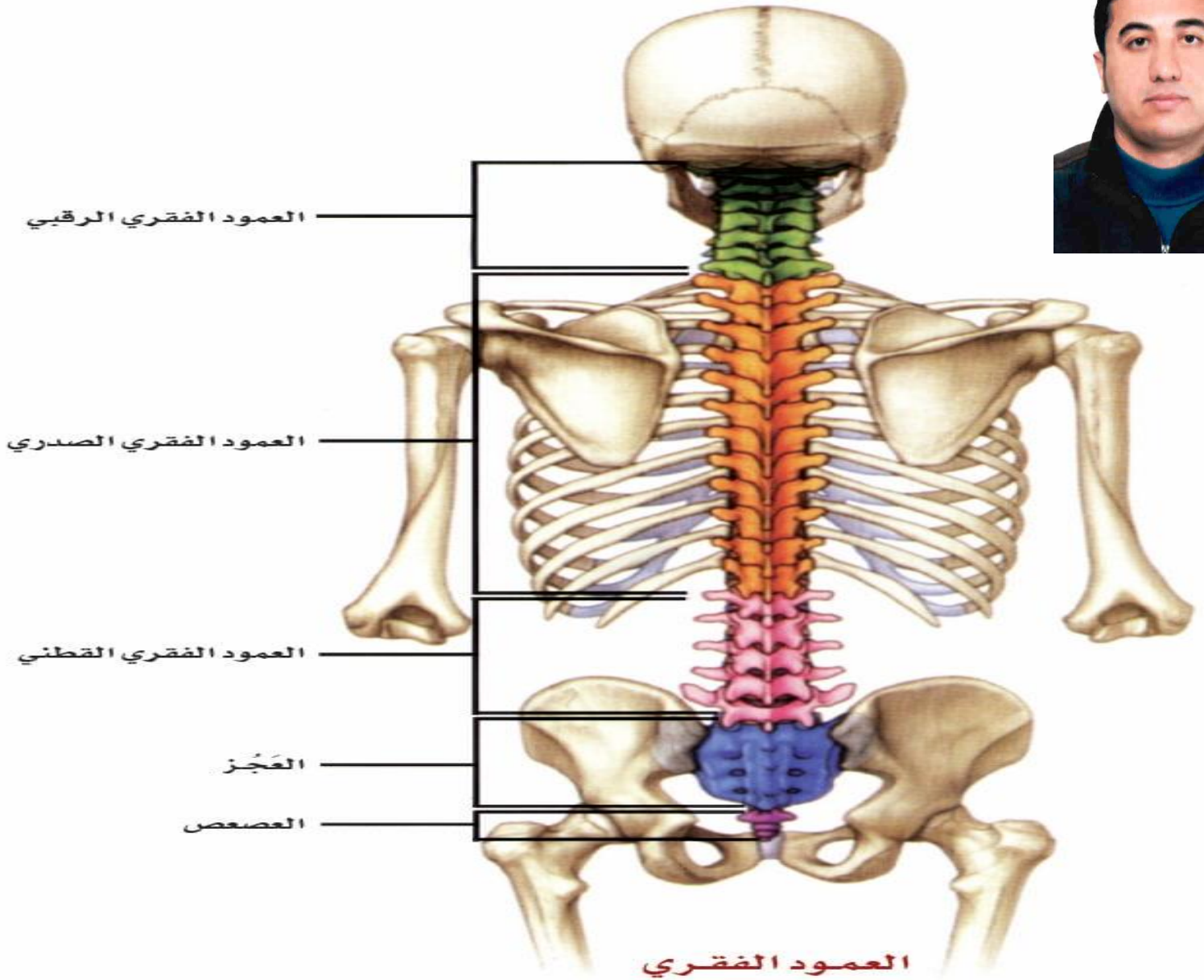
١. الفقرات الرقبية وعددها ٧ فقرات تشكل الانحناء الرقبي للعمود الفقري حيث تنحني هذه الفقرات بشكل طبيعي للأمام.

٢. الفقرات الظهرية أو العمود الفقري الصدري وعددها ١٢ فقرة ويكون انحناء العمود الفقري في هذه المنطقة نحو الخلف، وتتصل الفقرات الظهرية مع الأضلاع التي تكوّن القفص الصدري.

٣. الفقرات القطنية وعددها ٥ فقرات وهي تشكل منطقة أسفل الظهر ويكون انحناء العمود الفقري في هذه المنطقة للأمام بنفس جهة انحناء العمود الرقبي.

٤. العجز المكون من ٥ فقرات ملتحمة مع بعضها مشكلة قطعة عظمية واحدة مثلثية الشكل تقع تحت العمود الفقري القطني مباشرة.

٥. العصعص ويتكون من ٤ أو ٥ فقرات ملتحمة وهو القسم الانتهائي من العمود الفقري.



العمود الفقري

بيانات هامة للإتصال بصاحب

الموبايل نوكيا 130 –

دكتورمحمدمحمودوهيبه وعائلته :

دكتورمساعدباحث

/محمدمحمودمحمديوسف وهيبه

01210973074

أبى 01221868424

أمى 01206372319

أختى 01220781971

أخى 01288849405

دكتور عادل عبدالعزيز مدير العمل

01006646670

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس في العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جداً) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

بيان التقديرات الحاصل عليها الفتاة، مراحله بالكلية :-

الفرقة الأولى: دور: مايو عام: ١٩٩٨/١٩٩٩م

المادة	الفيزياء: العيوب	الميكانيكا: ديناميكا وسيوان	الكيمياء: العامة	التشريح والأنسجة	المستويات	الوراثة والخدمة	أحياء: عيوب	لغة إنجليزية	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	ممتاز	جيد//	ممتاز	جيد جداً
الدرجة	٣٥	٧٦	٨٢	٧٧	٧٤	٩٤	٢٣	٤٥	٥١٦

الفرقة الثانية: دور: مايو عام ١٩٩٩/٢٠٠٠م

المادة	تشريح الطيور ومكارن	الفسيولوجيا	طفيليات: عمومية	تغذية الحيوان والدواجن وأمراض سوء التغذية	سلوك الحيوان والدواجن وبر دائرتها	خاص الدم	التقدير العام
التقدير	جيد جداً	جيد جداً	ممتاز	جيد//	مقبول	مقبول	جيد جداً
الدرجة	٨١	٨٢	٩١	٧٢	٦٤	٣٠	٣٨٩

الفرقة الثالثة: دور: مايو عام: ٢٠٠٠/٢٠٠١م

المادة	باثولوجيا عام	مختبر باثولوجيا وطفيليات الماشية	الطفيليات	فارماكولوجيا	إنعاش حيواني	القضاء وإدارة برلم	فيزيولوجيا	التقدير العام
التقدير	مقبول	جيد جداً	جيد جداً	مقبول	جيد//	جيد//	مقبول	جيد//
الدرجة	٥٠	٨١	٧٨	٧١	٦٩	٧٤	٣٢	٤٥٥

الفرقة الرابعة: دور: مايو عام: ٢٠٠١/٢٠٠٢م

المادة	باثولوجيا تشريحي مرضي	أمراض باطنة	جراحة عامة وتخدير	باطنة (تناسلية)	طب شرعي	الرقابة الصحية على الألبان	باثولوجيا إكلينيكية	أمراض التناسل	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	جيد //	جيد//	جيد جداً	جيد//
الدرجة	٧٤	٦٩	٧٧	٧٦	٧٠	٧٣	٣٥	٤٠	٥١٤

الفرقة الخامسة: دور: مايو عام ٢٠٠٢/٢٠٠٣م

المادة	أمراض باطنة	جراحة عامة والتخدير	التوليد والتلقيح الصناعي	أمراض وجراحة	أمراض الطيور والأرانب	الرقابة الصحية على اللحوم ومنتجاتها	صحة الحيوان والدواجن والبيئة	الأمراض المشتركة	التقدير العام
التقدير	مقبول	جيد جداً	جيد//	جيد جداً	جيد//	جيد//	جيد//	ممتاز	جيد//
الدرجة	٦٤	٧٩	٦٩	٨٢	٧٣	٦٧	٦٦	٨٥	٥٨٥

وقد أعطيت له هذه الشهادة بناء على طلبه وتحت مسئوليته لتقديمها إلى من يهمه الأمر

تحريراً في: ٢٠١٨/١٠/٣٠م

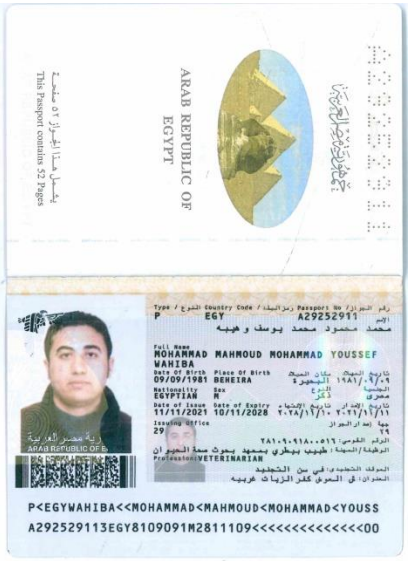
الموظف المختص: محمد الطالبة

وكيل الكلية لشئون التعليم والطلاب

عميد الكلية

((أ.م/ محمود محمد الدرديري))

((أ.م/ إسماعيل إسماعيل إبراهيم))



تتكون كل فقرة من جسم الفقرة وهو القسم الأمامي منها ومن بروزات عظمية عديدة ندعوها النواتئ ترتكز العضلات عليها، ويوجد في مركز كل فقرة حفرة محمية بالعظم المحيط بها، وتشكل الحفر مع بعضها قناة ضمن العمود الفقري هي القناة الشوكية التي يسكنها النخاع الشوكي.

يوجد بين كل فقرتين وسادة اسفنجية اسطوانية الشكل ندعوها القرص بين الفقرات (أو الديسك)، ويتكون هذا القرص من نواة جيلاتينية طرية ندعوها النواة اللبية تتوضع في مركز القرص، ويحيط بها حلقة ليفية قوية مكونة من شرائح متوضعة بشكل متحد المركز تماماً مثل شرائح البصل، ويعتبر القرص بين الفقرات مفصلاً يسمح بحركة العمود الفقري. وهناك ٢٣ قرصاً بين الفقرات يشكلون ثلث ارتفاع العمود الفقري ويخضعون لعمل مستمر، فهذه الأقراص هي التي تمتص الصدمات وتغيرات الضغط التي يخضع لها العمود الفقري، وتكون وظيفة الفقرات الصلبة تحويل هذه الضغوط إلى الأقراص بين الفقرات الملزمة بالتكيف مع الضغوط المختلفة، وفي الحقيقة يؤدي تعرض القرص إلى ضغط شديد إلى تسطح النواة اللبية بغية توزيع الضغط على أكبر مساحة ممكنة، وهذا يؤدي إلى تمدد الحلقة الليفية. وأثناء الراحة يحدث العكس حيث تشتد ألياف الحلقة الليفية ويستعيد القرص ارتفاعه الطبيعي.

بيانات ومعلومات طالب الزواج

بيانات شخصية :

- الإسم/ محمدمحمودمحمديوسف وهيبه
- العمل /مساعدباحث بمعهد بحوث الصحة الحيوانية
- المؤهل / ماجستير البكتيريا والمناعة والفطريات
- (عام 2021/5/19)
- العمر / أربعون عاما
- الحالة الإجتماعية / أعزب لم يسبق لى الإرتباط والزواج
- الخدمة العسكرية / تمت تأديتها
- الإقامة / شارع مجمع المدارس/البيت الأبيض المقابل لمحل ماكس جيم لكمال الأجسام/الدور الثانى ملكى/منزل الأستاذ محمودوهيبه /منطقة العوض/قرية الدلجمون /مركز كفرالزيات / محافظة الغربية
- رقم الموبايل / 01210973074
- البريد الالكتروني : m0hammed_yousef@yahoo.com
- الطول 180سم
- الوزن 100كيلوجرام
- لون البشرة أبيض
- الراتب حوالى 5000 جنيه شهريا
- المطلوب عروسة وزوجة مناسبة لى

بيانات وظيفية :

التعيين / بمعهد بحوث الصحة الحيوانية بالدقي / مصر/فرع طنطا كالتالي :-

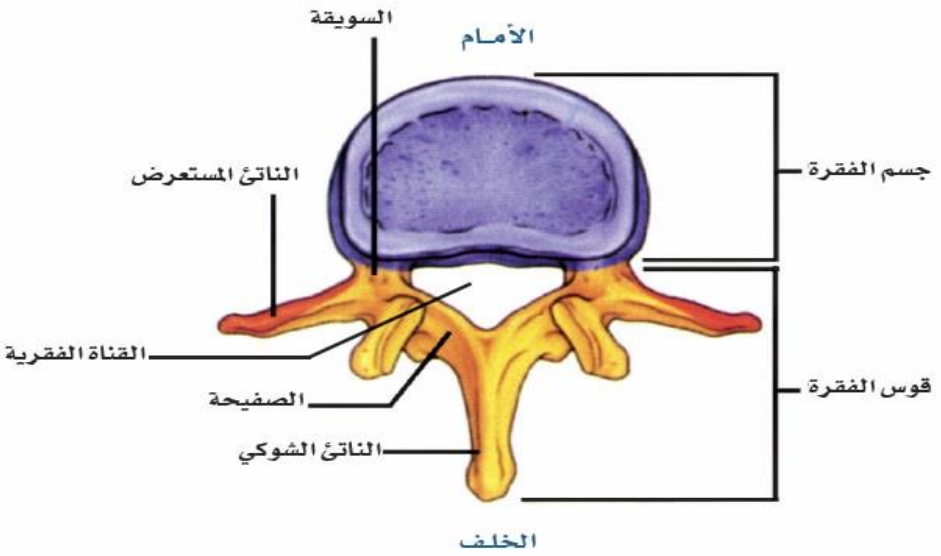
1- طبيب بيطري (من عام 2009إلي 2020)
2- مساعد باحث (من عام 2020/6 الي الوقت الجاري)
3- الحصول على درجة الماجستيرفى البكتيريا والمناعة والفطريات (بتاريخ 2021/5/19)
4- طبيب بيطرى فى الخدمة العسكرية الإلزامية فى الرقابة الصحية على الأغذية

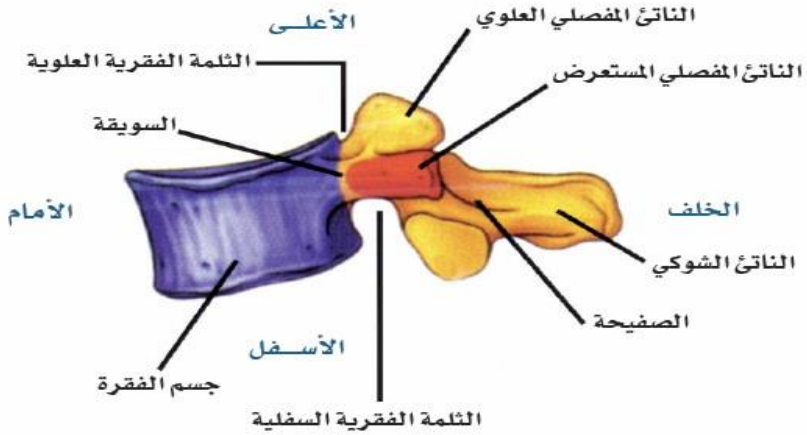
(اللحوم-الألبان ومنتجاتها)

المطلوب من معاليكم زوجة مناسبة

ولسيادتكم وافر التحية والتقديروالإحترام

متمنيا لسعادتكم مزيدا من التقدم والرقي





داء الفقار

دَاء الفَقَار أو تَنَكُّس الفَقار هو انتكاس العمود الفقري لأي سبب. بالمعنى الدقيق، يشير إلى هشاشة العظام في العمود الفقري، وهو تآكل وتمزق العمود الفقري المرتبط بتقدم العمر، وهو السبب الأكثر شيوعًا للإصابة بداء الفقار. تؤثر العملية التنكسية في هشاشة العظام بشكل رئيسي على الأجسام الفقرية، والثقب العصبي، ومفاصل الوجه (متلازمة الوجه). إذا كانت شديدة، فقد تسبب ضغطًا على النخاع الشوكي أو جذور الأعصاب مع اضطرابات حسية أو حركية لاحقة، مثل الألم والتنمل وعدم التوازن وضعف العضلات في الأطراف

عندما تقل المسافة بين فقرتين متجاورتين، قد يؤدي ضغط جذر العصب الخارج من الحبل الشوكي إلى اعتلال الجذور (اضطرابات حسية وحركية، مثل ألم شديد في الرقبة أو الكتف أو الذراع أو الظهر أو الساق، مصحوبًا بضعف العضلات).

أقل شيوعًا، قد يؤدي الضغط المباشر على النخاع الشوكي (عادةً في العمود الفقري العنقي) إلى اعتلال النخاع، الذي يتميز بضعف عالمي، وخلل في المشي، وفقدان التوازن، وفقدان السيطرة على الأمعاء أو المثانة. قد يعاني المريض من صدمات (تنمل) في اليدين والرجلين بسبب انضغاط العصب وقلة تدفق الدم. في حالة إصابة فقرات العنق، يتم تسميتها بداء الفقار الرقبية. يسمى داء الفقار أسفل الظهر داء الفقار القطني. المصطلح مشتق من اليونانية القديمة σπόνδυλος *spóndylos*، «فقرات - »، في الجمع «فقرة»، العمود الفقري».

جمهورية مصر العربية

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية **الطـــب البيـــطري**

أن / **محمـــد محمـــود محمـــد يوسف**

المولود فى **البحيرة** بتاريخ **١٩٨١م**

قد حصل على درجة **البكالوريوس فى العلوم الطبية البيطرية**

فى دور **مايو** عام **٢٠٠٣م** بتقدير **جيـــد جـداً**

مجموع تراكمى **٢٤٧٥ / ٣٣٠٠** النسبة المئوية **٧٥,٠٠%**

و قد اعتمد مجلس الكلية نتيجة إمتحانه فى **٢٠٠٣/٧/١٥م**

ومجلس الجامعة فى **٢٩ /٢٠٠٣/٧م** **بمقتضى قرار مجلس جامعة طنطا**

الموظف المسئول | المراجع | مدير شئون الطلاب | عميد الكلية

تحريراً فى **٢٠١٢/٥/٢٢م**

جمهورية مصر العربية

وزارة الداخلية

قطاع مصلحة الأحوال المدنية

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صورة قيد الميلاد

رقم القومى ٣٨١٠٩٠٩ ١٨ ٠٥١٦ ٠

بيانات المولود

محمد

الجنسيه :مصر

الديانه :مسلم

النوع :ذكر

محل الميلاد :البحيرة / مركز ابوحمص

تاريخ الميلاد :تسعة من سبتمبر

عام الف و تسعمائه و واحد و ثمانون

بيانات الأب

محمود محمد يوسف وهيبه

الديانه :مسلم

الجنسيه :مصر

بيانات الأم

ليلى محمد عبد المنعم

الديانه :مسلمة

الجنسيه :مصر

م . صحة : ابوحمص المختار رقم القيد :١٧٨٣

س .مدني : مركز ابوحمص ت .القيد :٣٠ /١٠ /١٩٨١

س . امد آر : سجل كفر الزيات ت .امد آر :٢٨ /٠٩ /٢٠١٧

رقم مسلسل ٢١٩٠٤٧٨٥٤٠

توقيع

تأكد من وجود طابع الأمومة والطفولة هلة ، جنيه والعلامة المائية ، نسر شعار الجمهورية ، وشهادة أحوال مدنية.

POLICE PRESS /2590630

((نموذج رقم ٢٥ س))

٣٥٤٣٣٧٣

إصدار ثانى

وزارة الدفاع
شهادة تأدية الخدمة العسكرية
بالقوات المسلحة بجمهورية مصر العربية

تشهد وزارة الدفاع

أن

رقم عسكرى: ٢٠٠٤٥٧٨٨٠١٩٦٦

درجة: ملازم مجند — اسم: محمد محمود محمد يوسف

الرقم القومى: ٢٨١٠٩٠٩١٨٠٠٥١٦ — سجل مدنى: مغر الزيات

محافظة: الغربية

قد انتهت مدة خدمته العسكرية
قوات الجيش
عن مدة خدمة حسنة قدرها

يوم ٥ شهر ١ سنة

بتاريخ يوم ٠١ شهر ٠٩ سنة ٢٠٠٥

بسبب نقله الى الاحتياط تنقضى مدة خدمته بالقوات الاحتياطية

يوم شهر سنة

وكانت درجة أخلاقه أثناء الخدمة قدوة حسنة

تحريرا بتاريخ ٢٠١٩/٠١/٢١

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

بيان مدة الخدمة العسكرية/ كود التعبئة ٥٩٠١٠١٠٠٣

البيان	يوم	شهر	سنة
تاريخ الالتحاق	٢٦	٠٧	٢٠٠٤
تاريخ انهاء الخدمة	٠١	٠٩	٢٠٠٥
اجمالي مدة الخدمة	٥	١	١
المدة الفائدة	٠	٠	٠
صافي مدة الخدمة	٥	١	١

من			إلى			المدة		
يوم	شهر	سنة	يوم	شهر	سنة	يوم	شهر	سنة

الموقف من الخدمة بالقوات الاحتياطية

يتم الرجوع لإدارة شئون ضباط القوات المسلحة لحين بلوغ سن التقاعد للرتبة

تعليمات ونصائح

١ - حافظ على هذه الشهادة سليمة وقدمها إلى كل جهة تطلبها منك.

٢ - الخدمة بالقوات الاحتياطية جزء من الخدمة العسكرية المفروضة عليك قانونا ويجب تأديتها كاملة.

٣ - عندما تستدعى للخدمة بالقوات الاحتياطية عليك التقدم إلى الجهة أو الوحدة التى ستحددها لك تعليمات الاستعداد فى التاريخ الذى يعين لذلك.

٤ - التخلف عن الحضور يوقعك تحت طائلة العقوبة التى قررها القانون لذلك.

٥ - اذا ما حدث ما يسمح لك المغادرة من الخدمة الاحتياطية تقدم إلى إدارة التجنيد والتعبئة لاتخاذ اللازم.

٦ - اذا فقدت منك هذه الشهادة سارع بالاتصال بأجهزة السجلات لاستخراج بدلا منها.

٧ - لا تضع نفسك تحت طائلة القانون سواء بالتعاقد أو العمل بالجيوش الأجنبية بالصفة المدنية أو العسكرية دون الحصول على تصديق مسبق من الجهات المختصة.

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

إدارة السجلات العسكرية - خدمة بمكاتب سجلات الغربية

نقابة الأطباء البيطريين
Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف
المهنة : طبيب بيطرى
رقم القيد : ٣١٦٧٩ / البحيرة
رقم الترخيص : ٣١٧٧٠
سارى حتى : ٢٠٢٠/٠١
الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام
د.حميدة الشيخ

اتحاد نقابات المهن الطبية
نقابة الأطباء البيطريين
www.egy-vet-synd.org

جامعة بنهـــا
مركز الشهادات الموحدة
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026

منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتـــاريخ : ١٩٨١/٩/٩
الجنســـية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكليـة : ٢٧/ ٤/ ٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥/ ٢٠٢١

الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥

المختص ماجد | رئيس القسم أ/أحمد البلاز | وكيل الكلية للدراسات العليا أ.د/ أمانى عبد الرحمن عباس | عميد الكلية

مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

(نموذج رقم ٦٠ ش ش)

جمهورية مصر العربية
وزارة الدفاع
إدارة شئون الضباط للقوات المسلحة
القيد : ٤٩٥٧
التاريخ: ٢٠٠٧/١٢/١٨ م

شهادة
خبرة لضابط إحتياط / مكلف

الى / م.أول احتياط غير مستدعى محمد محمود محمد يوسف

بناء على طلبكم المؤرخ فى ٢٠٠٧٠٣٠٠٤

بشأن إخطاركم بالأعمال التى مارستموها خلال فترة أستدعائكم بالقوات المسلحة

نفيدكم بأنكم قمتم بالأعمال الآتية:

الرقابة الصحية على الأغذية(اللحوم - الألبان ومنتجاتها) .

وذلك خلال الفترة من (٢٠٠٥/٠٩/٠١) الى (٢٠٠٦/١٢/٣١)

و القوات المسلحة تشكركم على خدمتكم و تتمنى لكم التوفيق .

لواء أ.ح / أحمد محمد أحمد نصر
مدير إدارة شئون الضباط للقوات المسلحة
بالتفويض التوقيع (.....)
عقيد / محمد ثروت حسان
رئيس فرع الشئون الشخصيه والخدمه الاجتماعيه

المضاعفات

المضاعفات نادرة ولكن الشديدة في هذا المرض هي قصور العمود الفقري.[١] يحدث هذا بسبب انسداد الشريان الفقري خلال مروره في الثقبة المستعرضة. تصبح مفاصل العمود الفقري صلبة في داء الفقار الرقبية. وهكذا تصبح الخلايا الغضروفية التي تحافظ على القرص محرومة من التغذية وتموت. قد تسبب النبتات العظمية الثانوية تضيقًا للأعصاب الشوكية، يظهر في شكل اعتلال الجذور.

الأسباب

ينتج داء الفقار عن سنوات من الضغط غير الطبيعي المستمر، الناتج عن خلع جزئي للمفصل، أو الإجهاد الناجم عن الرياضة، أو الصدمات الحادة أو المتكررة، أو الوضع السيئ، الذي يتم وضعه على الفقرات والديسك الموجودة بينها.

يتسبب الإجهاد غير الطبيعي في تكوين الجسم لعظام جديدة للتعويض عن توزيع الوزن الجديد. سيؤدي هذا الوزن غير الطبيعي الناتج عن إزاحة العظام إلى حدوث داء الفقار. يمكن أن تؤدي المواقف السيئة وفقدان الانحناءات الطبيعية للعمود الفقري إلى الإصابة بداء الفقار أيضًا. يمكن أن يصيب داء الفقار أي شخص في أي عمر؛ ومع ذلك، فإن كبار السن هم أكثر عرضة للإصابة.

التشخيص

تعد فحوصات التصوير بالرنين المغناطيسي والتصوير المقطعي المحوسب مفيدة في تشخيص الألم ولكنها ليست نهائية بشكل عام ويجب أخذها في الاعتبار مع الفحوصات البدنية والتاريخ.

العلاج

عادة ما يكون العلاج متحفظًا بطبيعته. تثقيف المريض حول تعديلات نمط الحياة، والعقاقير المضادة للالتهابات (NSAIDs) والعلاج الطبيعي، ورعاية تقويم العظام هي أشكال شائعة من الرعاية اليدوية التي تساعد في إدارة مثل هذه الحالات.[٥] قد تكون العلاجات البديلة الأخرى مثل التدليك وعلاج نقطة الاثر واليوجا والوخز بالإبر ذات فائدة محدودة. يتم إجراء الجراحة من حين لآخر

تتم الدعوة إلى الجراحة لعلاج اعتلال الجذور العنقي في المرضى الذين يعانون من آلام مستعصية أو أعراض تقدمية أو ضعف لا يتحسن مع العلاج المحافظ.

لا تزال المؤشرات الجراحية لداء الفقار العنقي (CSM) مع اعتلال النخاع مثيرة للجدل إلى حد ما ، ولكن «يوصي معظم الأطباء بالعلاج الجراحي بدلاً من العلاج المحافظ للاعتلال النخاعي المتوسط إلى الشديد»

قد يكون العلاج الطبيعي فعالاً لاستعادة نطاق الحركة والمرونة وتقوية القلب

ومع ذلك، فإن العلاج الطبيعي وعلاج العظام لا يمكن أن «يعالج» الانحطاط، ويرى بعض الناس أن الامتثال القوي لتعديل الوضعية ضروري لتحقيق أقصى فائدة من تخفيف الضغط والتعديلات وإعادة تأهيل المرونة.

ومع ذلك، فقد قيل أن سبب داء الفقار هو ببساطة الشيخوخة (على الرغم من أنه يمكن أن يكون موجودًا في أي عمر)، وغالبًا ما يمارس علاج تعديل الوضع من قبل أولئك الذين لديهم مصلحة مالية (مثل تعويض العمال)[٧] في إثبات أنه ناتج عن ظروف العمل والعادات الجسدية السيئة. فهم علم التشريح هو مفتاح الإدارة المحافظة لداء الفقار.

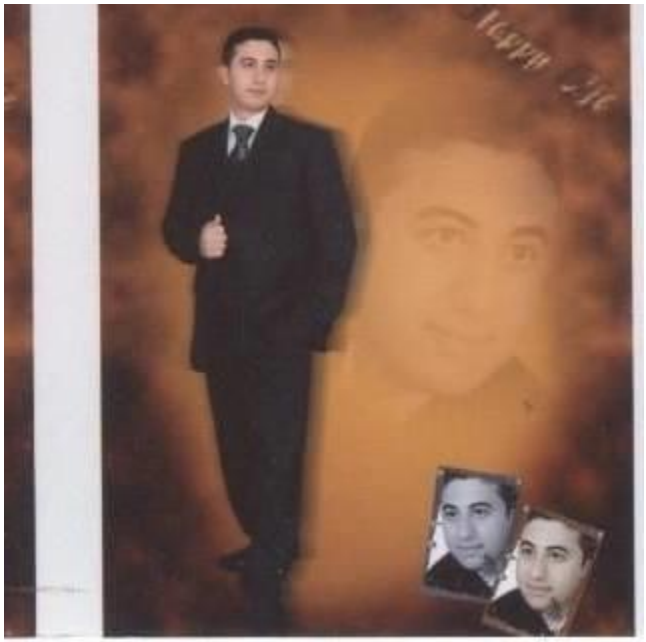
الجراحة

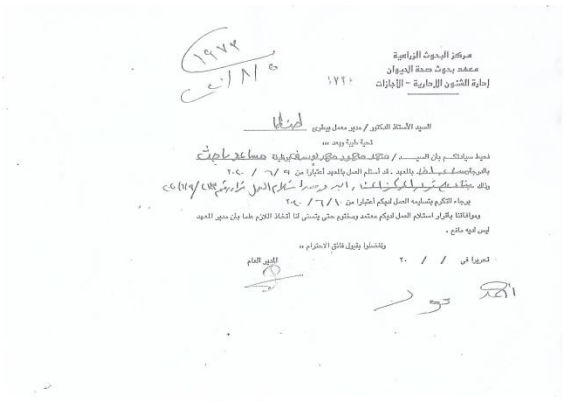
تهدف الإجراءات الجراحية الحالية المستخدمة في علاج داء الفقار إلى تخفيف علامات وأعراض المرض عن طريق تقليل الضغط في القناة الشوكية (جراحة تخفيف الضغط) و / أو عن طريق التحكم في حركة العمود الفقري (جراحة الدمج).

جراحة تخفيف الضغط: يمكن إجراء جراحة العمود الفقري من كلا المقاربة الأمامية والخلفية. يختلف النهج باختلاف الموقع وسبب ضغط الجذر. بشكل عام، تتم إزالة النباتات العظمية وأجزاء من القرص الفقري.

جراحة الدمج : تُجرى عندما يكون هناك دليل على عدم استقرار العمود الفقري أو سوء المحاذاة. يختلف استخدام الأجهزة (مثل البراغي السنية) في جراحات الاندماج عبر الدراسات.

فرط أوكسالات البول والداء الأوكسالى





جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة
بتاريخ : ٩/٩/ ١٩٨١
الجنسية : مصرى
الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٧/ ٤ /٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥ /٢٠٢١
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥
المختص
رئيس القسم
وكيل الكلية للدراسات العليا
عميد الكلية
ا/أحمد البار
أ.د/ أماني عبد الرحمن عباس
مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

نظرة عامة

يحدث فرط أوكسالات البول عندما يحتوي البول على قدر كبير من الأوكسالات. وتُعد الأوكسالات مادة كيميائية طبيعية في الجسم، وتتوفر أيضًا في أنواع طعام معينة. ولكن قد يسبب وجود قدر كبير من الأوكسالات في البول مشاكل خطيرة.

وقد ينتج فرط أوكسالات البول عن اضطراب وراثي (جيني)، أو عن مرض معوي، أو تناول الكثير من الطعام الغني بمادة الأوكسالات.

وتعتمد صحة الكلى طويلة الأمد على التشخيص المبكر لفرط أوكسالات البول وعلاجها العاجل.

يحدث داء الأوكسالي بعد الإصابة بفشل الكلى للأشخاص الذين لديهم أسباب أولية، ومعوية للإصابة بفرط أوكسالات البول، وتراكم الأوكسالات المتزايدة في الدم. مما يؤدي إلى وجود رواسب للأُوكْسالات منها في الأوعية الدموية، وعظام الجسم وأعضائة.

الأعراض

غالبًا ما يكون أول أعراض فرط أوكسالات البول تَكوُّن حصوات الكلى. وقد تشمل أعراض حصوات الكلى ما يلي:

ألم حاد أو مفاجئ في الظهر

ألم مستمر لا يتوقف في المنطقة الواقعة أسفل الضلوع في الظهر (الخاصرة)

ظهور دم في البول

حاجة ملحة إلى التبول المتكرر

ألم مصاحب للتبول

القشعريرة أو حمى

الأسباب

تحدث حالة فرط أوكسالات البول عندما يكون هناك كمية كبيرة من مادة تسمى الأوكسالات في البول. يوجد عدة أنواع من حالة فرط أوكسالات البول:

فرط أوكسالات البول الأولي. فرط أوكسالات البول الأولي يعتبر حالة نادرة وراثية (جينية) تتواجد منذ الولادة. في هذا النوع، لا يُصنّع الكبد ما يكفي من بروتين معين (إنزيم) يمنع الإفراط في إنتاج الأوكسالات، أو أن الإنزيم لا يعمل بشكل صحيح.

يتم التخلص من الأوكسالات الزائدة من خلال كليتيك، في البول. الأوكسالات الإضافية يمكن أن تتحد مع الكالسيوم لتكوين حصوات وبلورات الكلى، والتي يمكن أن تتلف الكلى وتتسبب في توقفها عن العمل (الفشل الكلوي).

تتشكل حصى الكلى مبكرًا وغالبًا ما تسبب أعراضًا خلال الطفولة أو المراهقة. بسبب الكميات الكبيرة المنتجة من الأوكسالات، تفشل الكليتان بحلول مطلع فترة البلوغ وحتي أواسطها لدى العديد من الأشخاص المصابين بفرط أوكسالات البول الأولي. ولكن الفشل الكلوي يمكن أن يحدث في مرحلة مبكرة، مثل مرحلة الرضاعة، في حين أن الآخرين ممن يعانون فرط أوكسالات البول الأولي لا يتطور أبدًا الأمر لديهم إلى الفشل الكلوي. حتى الآن، حدد الخبراء ثلاثة أسباب وراثية مختلفة لفرط أوكسالات البول الأولي.

الداء الأوكسالي. يحدث الداء الأوكسالي إذا كنت مصابًا بفرط أوكسالات البول الأولي وكليتاك تتدهوران. ولأن جسمك لم يعد قادرًا على التخلص من الأوكسالات الإضافية، فإنها تبدأ في التراكم — أولاً في الدم، ثم في عينيك والعظام والجلد والعضلات والأوعية الدموية والقلب وغيرهم من الأعضاء. هذا يمكن أن يسبب مشاكل متعددة.

فرط أوكسالات البول المعوي. العديد من الأمراض المعوية، بما في ذلك مرض كرون ومتلازمة الأمعاء القصيرة الناجمة عن الإجراءات الجراحية، تزيد من امتصاص الأوكسالات من الأطعمة، والذي يمكن أن يزيد من كمية الأوكسالات التي تُفرز في البول.

فرط أوكسالات البول المتعلق بتناول الأطعمة عالية الأوكسالات. تناول كميات كبيرة من الأطعمة التي تحتوي على مستويات عالية من الأوكسالات يمكن أن يزيد من خطر إصابتك بفرط أوكسالات البول أو حصوات الكلى. اطلب من طبيبك أو أخصائي التغذية قائمة بالأطعمة عالية الأوكسالات. تجَنُب الأطعمة عالية الأوكسالات مهمًا للغاية إذا كنت مصابًا بفرط أوكسالات البول المعوي.

المضاعفات

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١/ م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس فى العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جدا) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية الطب البيطري

أن / محمد محمود محمد يوسف

المولود فى البحيرة بتاريخ ١٩٨١م

قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية

فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً

مجموع تراكمى ٢٤٧٥/٣٣٠٠ النسبة المئوية ٧٥,٠٠%

وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م

ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا

الموظف المسئول مستاء المراجع مدير شئون الطلاب عميد الكلية

تحريراً فى ٢٠١٢/٥/٢٢م

نقابة الأطباء البيطريين

Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف

المهنة : طبيب بيطرى

رقم القيد : ٣١٦٧٩ / البحيرة

رقم الترخيص : ٣١٧٧٠

ساري حتى : ٢٠٢٠/٠١

الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام

د.حميدة الشيخ

يمكن أن يؤدي إهمال علاج فرط أوكسالات البول الأولي في النهاية إلى تلف الكلى. وقد تتوقف الكلى عن العمل مع مرور الوقت. وفي بعض الأفراد، تكون هذه العلامة الأولى على الإصابة بالمرض.

وتتضمن علامات وأعراض فشل الكلى ما يلي:

انخفاض إخراج البول أو انعدامه كليًا

الشعور بالمرض أو التعب العام

فقدان الشهية والغثيان والقيء

شحوب لون البشرة المرتبط بفقر الدم

تورم اليدين والقدمين

يمكن أن يؤدي الداء الأوكسالي إلى مضاعفات متنوعة خارج الكلى، والتي تتضمن أمراض العظم وفقر الدم وقُرح الجلد ومشكلات القلب والعين، وفي الأطفال، فشل النمو الطبيعي.

التشخيص :-

ويقوم الطبيب بإجراء فحص طبي شامل والذي يتضمن مناقشة حول التاريخ الطبي والنظام الغذائي. تشمل فحوصات تشخيص فرط أوكسالات البول ما يلي

فحوصات البول، لقياس الأوكسالات ومستويات نواتج الأيض الأخرى في البول

فحوصات ألم، لبيان وظيفة الكلى ومستويات الأوكسالات في الدم

تحليل الحصوات، لتحديد مكونات حصوات الكلى التي تخرج مع البول أو بالإزالة الجراحية

الأشعة السينية على الكلى أو الموجات فوق الصوتية أو الفحص بالتصوير المقطعي المحوسب، بحثًا عن وجود أيّ حصوات في الكلى أو ترسبات أوكسالات الكالسيوم

وبعد الفحص الأول، قد ينصح الطبيب بالمزيد من الفحوصات لتأكيد التشخيص ومعرفة ما إذا كان المرض أصاب أجزاء أخرى من الجسم. قد تشمل تلك الاختبارات:

فحص الحمض النووي بحثًا عن الأسباب الوراثية (فرط أوكسالات البول الأولي)

خزعة الكلى بحثًا عن ترسبات الأوكسلات

مخطط صدى القلب لفحص ترسبات الأوكسلات في القلب

فحص العين لفحص ترسبات الأوكسالات في العين

خزعة نخاع العظم لفحص ترسبات الأوكسلات في العظام

خزعة الكبد لمعرفة حالات نقص الإنزيمات، حيث تلزم فقط في حالات نادرة عندما لا تكشف الفحوصات الوراثية عن سبب فرط أوكسالات البول

في حالة تشخيص إصابة المريض بفرط أوكسالات البول الأولي، يكون الأشقاء معرضين لخطر الإصابة بالمرض وينبغي خضوعهم للفحوصات. في حالة إصابة الأبناء بفرط أوكسالات البول الأولي، يمكن مراعاة إجراء الفحوصات الوراثية في حالة التخطيط لإنجاب مزيد من الأطفال. ويمكن للاستشاريين الوراثيين الطبيين المتمرسين أن يساعدوا في تقديم التوجيه بشأن القرارات والفحوصات.

العلاج:-

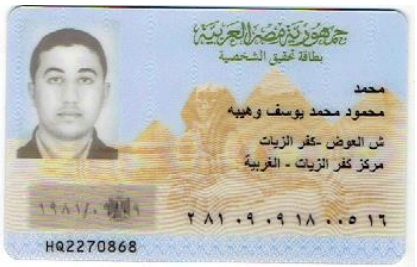
يعتمد العلاج على نوع مرض فرط أوكسالات البول وأعراضه وحدته ومدى جودة استجابتك للعلاج.

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

يمكن أن تكون جرعات فيتامين بى ستة فعالة في تقليل الأوكسالات في البول لدى بعض الأشخاص المصابين بفرط أوكسالات البول الأول.

تساعد مستحضرات السترات الفموية على منع تكوين بلورات أوكسالات الكالسيوم.

تناوُل كميات كبيرة من السوائل. إذا كانت الكليتان لا تزالان تعملان بصورة طبيعية، فمن المحتمل أن ينصحك طبيبك بشرب المزيد من الماء أو غيره من السوائل. إذ يعمل ذلك على غسيل الكلى ويمنع تراكم بلورات الأوكسالات ويساعد على منع تكوُّن حصوات الكلى.



التغيرات في النظام الغذائي. بوجه عام، تزداد أهمية العناية بنظامك الغذائي إذا كنت تعاني من فرط أوكسالات البول المعوي أو الغذائي. قد يوصي طبيبك بإدخال تغييرات إلى نظامك الغذائي، مثل تقليل الأطعمة التي تحتوي على نسبة عالية من الأوكسالات والحد من تناوُل الملح وتقليل البروتين الحيواني والسكر (شراب الذرة عالي الفركتوز). ويمكن أن تساعد مثل هذه التغييرات الغذائية على خفض مستويات الأوكسالات في البول. لكن قد لا تكون القيود الغذائية فعالة لدى جميع الأشخاص المصابين بفرط أوكسالات البول الأولي. اتبع توصيات طبيبك.

الأطعمة التي تحتوي على كميات كبيرة من الأكسالات

السبانخ.

اللوز.

الشمندر واللفت.

التوت.

زبدة الجوز.

المكسرات والكاجو.

الشوكولاتة والكاكاو.

ما أسباب تكون أوكسالات الكالسيوم؟

عدم الحصول على كمية كافية من السوائل. الإفراط في تناول نظام غذائي غني بالأوكسالات، أو البروتين، أو الملح. فرط نشاط جارات الغدة الدرقية. مرض التهاب الأمعاء.

السيطرة على حصوات الكلى

تنتشر حالات حصوات الكلى لدى المصابين بفرط أوكسالات البول ولكن لا تحتاج إلى العلاج دائمًا. إذا كانت الحصوات الكبيرة بالكلى تسبب الألم أو تعوق تدفق البول، فقد يحتاج المريض إلى إزالتها أو تفتيتها لتخرج مع البول.

الغسيل والزراعة

وفقًا لدرجة خطورة حالة فرط أوكسالات البول التي تعاني منها، ربما تصاب في نهاية المطاف بفقدان وظائف الكلى. ربما يكون غسيل الكلى مفيدًا بصورة مؤقتة، ولكنه لا يتوافق مع كمية الأوكسالات التي يتم إنتاجها. ربما تؤدي زراعة الكلى أو زراعة الكلى والكبد إلى التعافي من أنواع وراثية معينة من فرط أوكسالات البول (فرط أوكسالات البول من النوع الأول).

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

What is PCR (polymerase chain reaction)?
PCR is a technique used in the lab to make millions of copies of a particular section of DNA. It was first developed in the 1980s.

What is PCR?

The polymerase chain reaction (PCR) was originally developed in 1983 by the American biochemist Kary Mullis. He was awarded the Nobel Prize in Chemistry in 1993 for his pioneering work.

PCR is used in molecular biology to make many copies of (amplify) small sections of DNA? or a gene?.

Using PCR it is possible to generate thousands to millions of copies of a particular section of DNA from a very small amount of DNA.

PCR is a common tool used in medical and biological research labs. It is used in the early stages of processing DNA for sequencing?, for detecting the presence or absence of a gene to help identify pathogens? during infection, and when generating forensic DNA profiles from tiny samples of DNA.

How does PCR work?

The principles behind every PCR, whatever the sample of DNA, are the same. Five core 'ingredients' are required to set up a PCR. We will explain exactly what each of these do as we go along. These are:

- the DNA template to be copied
- primers, short stretches of DNA that initiate the PCR reaction, designed to bind to either side of the section of DNA you want to copy

DNA nucleotide bases? (also known as dNTPs). DNA bases (A, C, G and T) are the building blocks of DNA and are needed to construct the new strand of DNA

Taq polymerase enzyme? to add in the new DNA bases

buffer to ensure the right conditions for the reaction.

PCR involves a process of heating and cooling called thermal cycling which is carried out by machine.

There are three main stages:

Denaturing – when the double-stranded template DNA is heated to separate it into two single strands.

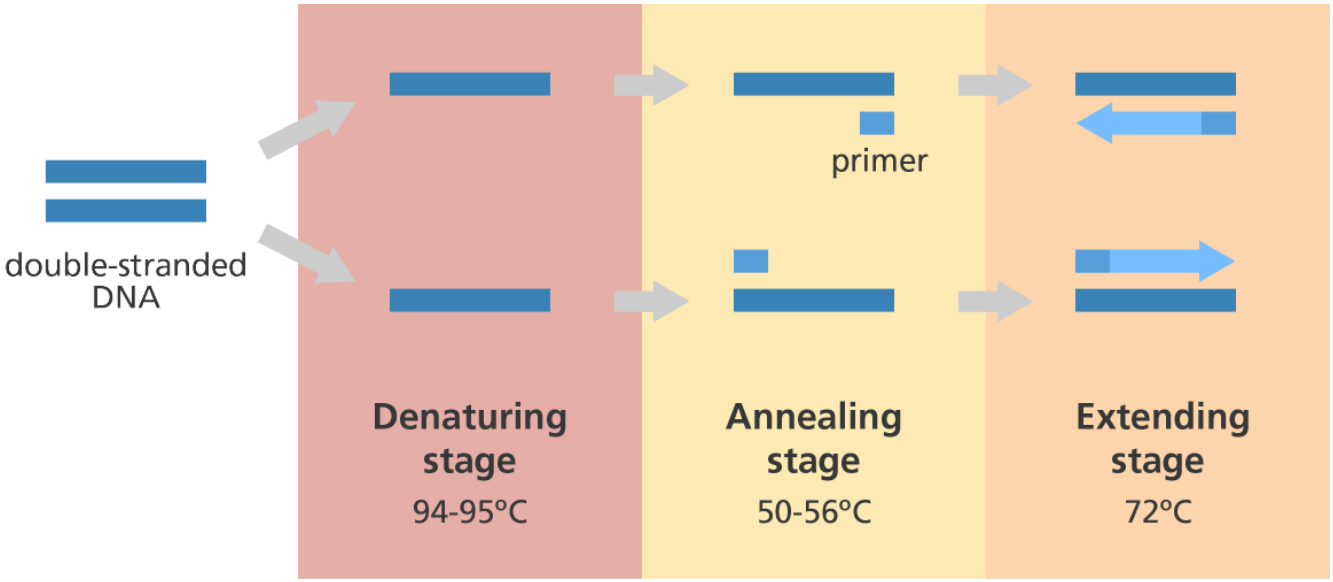
Annealing – when the temperature is lowered to enable the DNA primers to attach to the template DNA.

Extending – when the temperature is raised and the new strand of DNA is made by the Taq polymerase enzyme.

These three stages are repeated 20-40 times, doubling the number of DNA copies each time.

A complete PCR reaction can be performed in a few hours, or even less than an hour with certain high-speed machines.

After PCR has been completed, a method called electrophoresis can be used to check the quantity and size of the DNA fragments produced.



.(Illustration showing the main steps in the polymerase chain reaction (PCR)
Image credit: Genome Research Limited

Clostridia

Species:

Clostridium tetani
Clostridium botulinum
Clostridium perferingens (CL.welchii)
Clostridium septicum **and** ***Clostridium chauvoei***
Clostridium novyi (CL.oedematiens)

Definition:

This genus are spore forming rods which are anaerobic, some of them are pathigenic for man and animals.

The Clostridial group grows only in the absence of free oxygen (O_2) due to its lack of catalase and peroxidase enzymes which are necessary to split H_2O_2as end metabolites where H_2O_2 are toxic and harmful to the clostridium group. While free oxygen itself was not harmful to clostridium

General characters of Clostridial group :

Size and shape:

The members of clostridial group are bacilli with variable dimensions measuring (0.4 – 1.2µ × 3-8µ) in length. They are usually plump in shape with round ends, but some are long and slender rods.

Sporulation:

All of members are sporulated with rhe exception of *Cl.perfringens* spores have variable position and which may be central, sub-terminal or terminal.

Motility:

Most species are motile with pertrichous flegella except *Clostridium welchii* which is non motile.

Staining:

All members are gram positive mainly in young cultures.

Capsule:

Most members are non capsulated except *Cl. perferingens*.

Toxin production:

All pathogenic species give one or more types of exotoxins.

CLOSTRIDIUM WELCHII (CLOSTRIDIUM PERFRINGENS):

This group of organism are most commonly distributed in the intestinal tract of man and animals, there are 6 toxogenic typed (A,B,C,D,E,F) and differentiated according to the types of toxin produced. It causes different disease in man and animals.

Morphology:

It is gram positive and straight with parallel sides and rounded ends, spores are rarely seen. The organism are non motile and it is capsulated.

Cultural characters:

Clostridium welchii is not strict anaerobe; its surface colonies are circular, convex, semi translucent, smooth and white and entire edge, there is no tendency of spreading over the surface of the medium.

On horse blood agar: many strains produce double zones of haemolysis due to production of two toxins (theta and alpha) and absence of haemolysis is a characteristics of food poisoning strains of *Clostridium welchii* .

On heated blood agar: few strains produce evidence of slight proteolytic activity.

On egg-yolk agar: the organism produces opalescence which is inhibited by the specific *Cl. Welchii* antitoxin (Nagler reaction).

Nagler reaction (lecithinase activity)

Several clostridia are able to produce opalescence in both human serum and egg yolk media, due to the production of lecithinase which cause visible egg yolk media, due to the production of lecithinase which cause visible precipitates in these media.

The reaction is demonstrated with alpha (α) toxin of *Cl. Welchii* and is specifically inhibited by *Cl.welchii* anti toxin.

Nagler reaction has also been used for presumptive identification of *Cl.welchii* in direct plate culture when one half of the egg yolk medium inoculated with 2 or 3 drops of stander antitoxin and allowed to dry.

The whole plate is then inoculated with *Cl.welchii* starting with the half free from the antitoxin. On the half containing no antitoxin, *Cl. welchii* colonies shows surrounding zone of opacity i.e. Nagler reaction, while colonies, shows no change on the antitoxin half.

The Nagler reaction of the Clostridium members:

Organism produce opalescence	**Opalescence inhibited by *Cl. welchii* (alpha antitoxin)**
Cl. welchii (A-F)	+
Cl. Botulinum	-
Cl.oedematiens Type: A B D	 - - -

Biochemical Reactions:

In cooked meat media, the growth is evident in afew hours and the meat particles changes to pink, i.e. due to its saccharolytic activity.

All types ferment glucose, maltose, fructose, sucrose and liquefy gelatin.

Litmus milk is coagulated and acid and gas is produced giving stormy fermentation as in picture of test tube. H_2S is positive, indol negative, MR negative, VP negative and catalase negative.

Resistance and viability :

The vegetative form of the organism is not resistant to heat or chemicals, in spore form it is quite heat resistant, not being killed for 10 minute at 120 °C.

Toxins of *Clostridium welchii*:

The types of *Cl. Welchii* differ in the combination of various toxic and enzymic factors they produce. Several of these factors have haemolytic , lethal or necrotizing properties and others have enzymatic activity against biological substrate . the types of *Cl. Welchii* can be differentiated on the bases of four major lethal toxins (alpha, beta, epsilon and iota).beside to

these four major toxins there are 8 minor toxins (theta ,gamma, mu, nu, delta, kappa, leta and lambda).

Major toxin:

Alpha toxin: (α)

It is produced by all types of *Cl. welchii*, it is lethal and necrotizing , this toxin is an enzyme lecithinase C, and in presence of calcium ions it splits lipoprotein opalescence . The reaction can be inhibited by specific antiserum.

Beta toxin :(ß)

Produced by types B and C it is lethal and necrotizing.

Epsilon toxin:

It is produced by types B and D strains as a protoxin and therefore it needs a proteolytic enzyme e.g. pepsin or trypsin to be activated it is lethal and necrotizing, its activity depends also beside the proteolytic enzyme in alkaline medium .

Iota toxin:

Only type E produces this toxin as a protoxin so it needs pre-enzyme to be activated.

Minor toxins of *Cl. Welchii* :

Theta toxin:

It is an oxygen labile haemolysin , it is produced by all strains of *Cl. welchii* from A-F. It is lethal and hemolytic toxin.

Gamma toxin: it is lethal toxin.

Delta toxin:

It is lethal toxin and haemolytic for red cells of sheep, goat ,pigs and cattle.

Kappa toxin: it is collagenase which attacks native collagen and may cause softening of muscle connective tissues.

Mu toxin : It is a hyalurinidase

Nu toxin: It is deoxyribonuclease enzyme

Distribution of major toxins in different types of *Cl. welchii* and disease produced.

Cl. welchii types	Major toxin produced	Disease produced
A	Alpha	Gas gangrene in man and animals
B	Alpha, beta, epsilon	Lamb dysentery
C	Alpha, beta	Struck in sheep
D	Alpha, epsilon	Pulpy kidney disease in sheep
E	Alpha, iota	Enterotoxemia in calves and lambs

Typing of *Cl.welchii* strains

Clostridium welchii strains could be typed either by:

Neutralization test :

It is test of *Cl.welchii* was prepared and part (0.3) is put in 6 test tubes. The first tube left as control and antiserum (0.1) of types A.B.C.D and E was added to other test tubes respectively.

The tubes incubated at 37°C for 1hr. then 0.1 ml of each tube injected intra-venous in white mice and the deaths recorded after 24 hr.

The groups of mice which remain alive represent the type of *Cl. welchii*. i.e. if group pf mice injected each toxin (B) remain alive so type of *Cl. welchii* is type B and the first group must be died.

N.B. In neutralization test , antitoxin must be added to the toxin and not the reverse.

Dermonectrotic test:

Toxin of *Cl. welchii* was also prepared and antisera of types A, B, C,D and E are added. Then 0.1 ml of the toxin as it is or from the mixture (toxin –antitoxin) is injected intradermally on the back of guinea pigs after

removing the hair then left 24 hours and the result indicated by the presence of necrosis at the site of injections.

Nagler reaction or lecithinase activity:

Laboratory diagnosis of Cl. welchii:

Isolation and identification of the causative organism.

lecithinase activity or Nagler reaction on egg yolk media.

Demonstration of the type of strain and the toxin present in the intestinal content,

Identification and typing was applied by neutralization test using specific antitoxic sera by animal inoculation.

Laboratory diagnosis of Cl. welchii:

Isolation and identification of the causative organism.

lecithinase activity or Nagler reaction on egg yolk media.

Demonstration of the type of strain and the toxin present in the intestinal content,

Identification and typing was applied by neutralization test using specific antitoxic sera by animal inoculation.

GENUS: ESHCERICHIA
SPECIES: ESCERICHIA COLI (E.Coli)
Synonym: BACTERIUM COLI (E.Coli)

Definition: Escherichia coli is a normal inhabitant of the lower part of intestine of all warm blooded animals.

It is rarely present in stomach and in few numbers in the anterior part of intestine. Carnivores and omnivores usually harbor E.coli in greater number than herbivore.

Under certain conditions of stress and environmental factors may undergo to be potential pathogenic causing sever diseased conditions.

Specific diseases caused by enteropathogenic E.coli in different animal species:

Species	Disease	Symptoms
Childrens	Coli septicemia	**Diarrhea , death**
Newly born animals	Colibacillosis, white or calf scour	**Septicemia and rapid death**

Microscopic characters :

E.coli is coccobacilli to long rods (.6 x1.3M), arranged singly or in short chains especially in mucoid colonies where it had a slime layer of polysaccharides . it is motile and non – sporulated .

Cultural characters :

(Nutrient broth-Nutrient agar-Blood agar-MacConkey agar)

-In nutrient broth growth is rapid , gives regular turbidity within 12-18 hours at 37 C with heavy powdery sediment after 24 hours .

-On nutrient agar growth

1-smooth surface colonies occurs after 24 hours,

-The colonies are grayish in color , circular , convex 2-3mm in diameter .

 2- mucoid colonies appear due to the presence of slime layer and larger in size than smooth one .

-On blood agar the surface colonies are larger some are hemolytic and others are non-hemolytic

-On macConkey agar it gives round pink colonies, medium sized opaque colonies i.e lactose fermenter

(Viability And Resistance) :

-It may survive for weeks or months in contaminated water, dwellings in the animal houses in the presence of mucous or feces . Some strains resist dryness.

-It is susceptible to physical and chemical agents. E.coli is killed by heating at (55-60 C) for 30 minutes, within 10 minutes by autoclaving.

The Antigenic Structures Of E.coli include three antigens : 1-somatic antigens 2- capsular antigens 3- flagellar antigens . Detected by serological diagnosis

1-Somatic antigens (146 O- antigens)
2-Capsular antigens (91 K –antigens)
3- Flagellar antigens (49 H- antigens) :

1-Somatic Antigens : (146 O- antigens) :

E. coli strains have 146 O-antigens in their body . Each strain carries only one type of O-antigen . They are composed chemically of polysaccharide–phospholipids –protein complex. The O –antigens are designated by numbers as O1, O2, O3,O4 ,O5 – O 145 etc .

The O – antigens are heat stables.

2- Capsular antigens (91 K –antigens) :

These 91 k-antigens occur as a slime layer or microcapsule on most enteropathogenic E.coli strains.

Chemically they are composed of poly saccharides . k-antigens prevent agglutination between O-antigens and their specific homologous antisera.

The K – antigens are differentiated into

3 types according to their thermo stability as follows :

L-antigen : heat labile

A- antigen : heat stable

B- antigen : lipopoly saccharide destroyed by autoclaving

The K-antigen is very important in serotyping of E.coli as well as in the mechanism of pathogenesis as K 88 and K 99 .

These K 88 and K 99 mediate adhesion of the organism to the microvilli of the intestinal epithelial cells and enhance colonization .

3-Flagellar antigen (49 H-antigens) : There are 49 H-antigens which is composed of protein . frequently it is poorly developed .

It has weak role in serotyping of E.coli.

-Serotyping Identification :

For serotyping of E. coli , identification of O and K antigens is applied by slide agglutination test to determine its antigenic structures as O20/B6 , O55/B5 , O88/B4 , O111/B4 .

Toxins of E.coli (E.coli products) :

1-Enterotoxin 2- colicins

1-Enterotoxin:

This substances is produced by entero pathogenic strains of E. coli , and causes diarrhea in man and animals . Injection of E .coli enterotoxins into the ligated intestinal loops of rabbits induced swelling , oedema and accumulation of exudate in the injected loop. The enterotoxins are not produced by all E.coli strains but only the enteropathogenic.

E.coli enterotoxin is composed of two fractions:

a- Heat stable fraction.
b-Heat labile fraction.

a-Heat stable fraction : it is exteracellular in the culture . it is resistant to heating for 60 C but destroyed by autoclaving .
b- Heat labile fraction: it is inactivating by heating at 60 C for 30 minutes it is antigenic and occurred within bacterial cells . it could be neutralized by specific antisera .

2- Colicins :

It is E.coli products and composed chemically of protien . it has bactericidal activitiy . colicins productions is very important in differentiation of E.coli strains into :

Colicins positive E.coli.

Colicins negative E.coli.

Laboratory diagnosis:

- The isolation of E.coli may be of no value as E.coli are normal inhabitant of the intestinal tract, unless other seven factors are considered including:

1-Detailed history of the case, symptoms and lesions with differential bacteriological diagnosis on the other causative agents of digestive disorders .

2-Purity of the growth of E.coli on the primary cultures note the growth of lactose fermenting colonies on MacConkey agar .

3-Site of isolation from various organs especially from the parenchymatous organs as liver , spleen , mesenteric lymph nodes and lung tissues indicating colibacillosis.

4- Biochemically identification of isolated suspected E.coli the suspected E.coli colonies as lactose fermenter on macConkey media can be tested biochemically And must be differentiated from the other lactose – fermenter species by the following reactions (Indol , Methyle Red , VP , Citrate , Urea Hydrolysis , H2S, Motility , KCN) (Voges proskauer)(potassium cyanide)

E.coli are motile and Indol and Methyl Red Positives Only .

5-Serotyping of E. coli if the isolating colonies, are biochemically typical of E.coli, Serotyping must be performed using polyvalent O, K anti-sera to determine the antigenic structure of the isolated strain of E.coli.

6-Final diagnosis depends mainly upon the isolation of E.coli from various fresh organs in case of septicemia of the same antigenic structure.

7-It is very essential that the same serotype of E.coli must be isolated from feces or intestinal content as well as the internal organs of the same case .

Morphological character and staining of E.Coli

ساعات معتمدة ملف التسمم الغذائى دكتوراة مادةالبكتيريا

1-gram reaction gram negative coccobacilli – violet in color

2-shape coccobacilli to long rods singly or short chains

3-motility by peritrichous flagella

4-capsule - capsulated with slime layer

5-spore formation – non sporulated

Cultural character needs:

Oxygen : facultative anaerobic

Temperature : 37 C cellius degree

PH : 7.2

Incubation periods : after 24 hours

Selective media :

1-Eosin methylene blue agar (EMB) : green metallic colony

2-violet red bile agar (VBRA) : pink colony

Lactose fermentation : positive , H2S : negative , citrate utilization : negative , , oxidase : negative , catalase : positive , potassium cyanide : negative , vogus prouskour : negative , indol : positive , methyle red : positive . urea test : negative .

TOXINS OF ESCHERICHIA COLI :

1-ENTERO TOXINS

2-COLICINS

3-VEROTOXINS (SHIGA TOXINS): RESPONSIBLE ON BLOODY DIARRHEA

SURVIVAL AND RESISTANCE :

KILLED BY ANTIBIOTICS

PATHOGENESITY :

E.COLI CLASSIFIED IN TO SIX PATHOTYPES :

ENTEROPATHOGENIC E.C (EPEC)

ENTEROINVASIVE E.COLI (EIEC)

ENTEROHEMOLYTIC E.COLI (EHEC)

ENTEROTOXIGENIC E.COLI (ETEC)

ENTERO AGGREGATIVE E.COLI (EAgEC)

ENTEROUROPATHOGENIC E.COL I (EUEC)

PATHOGENICITY OF ESCHERICHIA COLI ON THE HUMAN AND ANIMALS AND POULTRY :

1-HUMAN FOOD POISONING WITH DIARRHEA , INFLAMMATION OF URINARY BLADDER

2-COWS AND BUFFALOS ONE OF MASTITIC CAUSE OR CAUSES OF MASTITIES AND COLIBACILLOSIS ESPECIALY IN NEWLY BORN CALVES

3-POULTRY CAUSING COLIBACILLOSIS AND CRD CHRONIC RESPIRATORY DISEASES WHICH ARE AMIXTURE BETWEEN E.COLI AND MYCOPLASMA

DIFFER BETWEEN FECAL FORM AND NON FECAL FORM :

BY USING EIJKMAN TEST :

IT IS DEPENDANT ON THE ABILITY OF BACTERIA TO GROW ON MACCONKEY AGAR 44 C CELLIUS DEGREE :

IF IT IS ABLE TO GROW ON THIS TEMPRATURE IT IS FECAL FORM E.COLI

IF IS NOT ABLE TO GROW ON THIS DEGREE IT IS NOT FECAL FORM E.COLI

LABORATORY DIAGNOSIS :

SAMPLING :

1-MASTITIS : MILK SAMPLE

2-DIARRHEA : FECES

3- INFLAMMATIONS OF URINARY TRACT : URINE

4-SAMPLE FROM VOMITION IF PRESENT

Salmonella

- Comprise a large group of serotypes (2700)

- World wide & infect a wide variety of hosts

As man , animals & poultry

- Salmonella are typical intestinal pathogens

& contaminate the environment by the organism in the feces and transfer infection to other

Some species are host spcific

species	group	host	disease
S.Paratyphi A	A	Man	Paratyphoid A fever
S.Paratyphi B	B	Man	Paratyphoid B fever
S.Abortus equi		Equine	Equine abortion & infertility
S.Abortus ovis		Ovines	Ovine abortion
S.cholera suis	C	swine	Piglet typhus Infectious enteritis
S.typhi	D	man	Human typhoid fever
S.gallinarum		poultry	Pullorum disease

salmonella

Genus : Salmonella

Species : S. typhi

S. typhimurium

S. enteriditis

S. gallinarum-pullorum

S. dublin

S. infantis

S. derby

S. agona

S. panama

S. heidlberg

morphology

- Cocco-bacilli
- Gram -ve
- Medium sized rods
- Actively motile expect S.gallinarum and pullorum
- Many species developed fimbria
- Non spore forming
- Non capsulated

Cultural characters

- **Faculatative anearobes . Temrpature 37C PH 7.2 incubation time 48 hours**
- **Grow in ordinary media**
- **In nutrient broth** **nutrient agar**

Turbidity , without Pellicle formation

round colonies, smooth, convex with grayish colour

S. gallinarum & S. abortus ovis

small, dew drop like colonies after 48 hours incubation

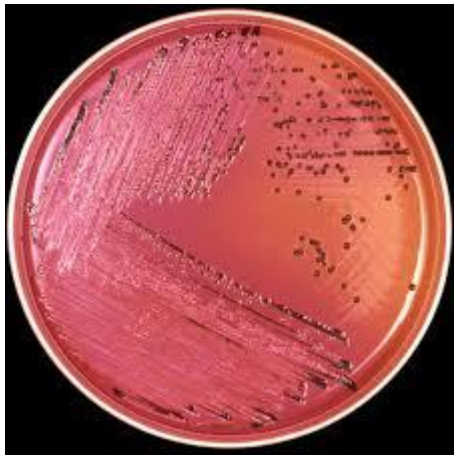
- **Onto blood agar ⟶ the colonies relatively large , non haemolytic**
- **Onto MacConkey agar pale in colour (non lactose fermenter)**

Selective media of Salmonella

There two selective media of salmonella

1- XLD (Xylose Lysin Deoxscholte) agar : colonies appear pink calour with black center due to salmonella produce H2S

2- S.S agar (Salmonella Shigella Agar) : give yellow coloniesn



Enrichment media

There is a more complex problem for salmonella isolation due to their presence in the intestinal tract

Examples :

- Tetrathionate brilliant green broth

- Selenite F broth

by growing the fecal samples or intestinal contents on the enriched media for 18 -24 hours to inhibit the growth of the other contaminants as E.coli , proteus

Biochemical reactions

- Lactose -ve
- Indole -ve
- MR +ve
- VP -ve
- Citrate +ve
- Urease -ve
- H2S +ve

Viability & resistance

- Salmonellae easily killed at 60ºC for 20 min.

- Quickly destroyed by the common chemical disinfectant

- **Identification of salmonella species depends mainly on the serotyping according to**

Kaufman & White scheme of the antigenic structure of salmonella

Antigenic structures

- **Somatic antigens O-antigen**

- **Present in the cell wall**

- **Lipopolysaccharide-protein complex**

- **Designated by arabic numbers from 1 – 65**

- **The majority of salmonallae posses more than one of somatic antigens (3-4) on their surface**

- **Flagellar & fimbrial antigens (H- antigens)**

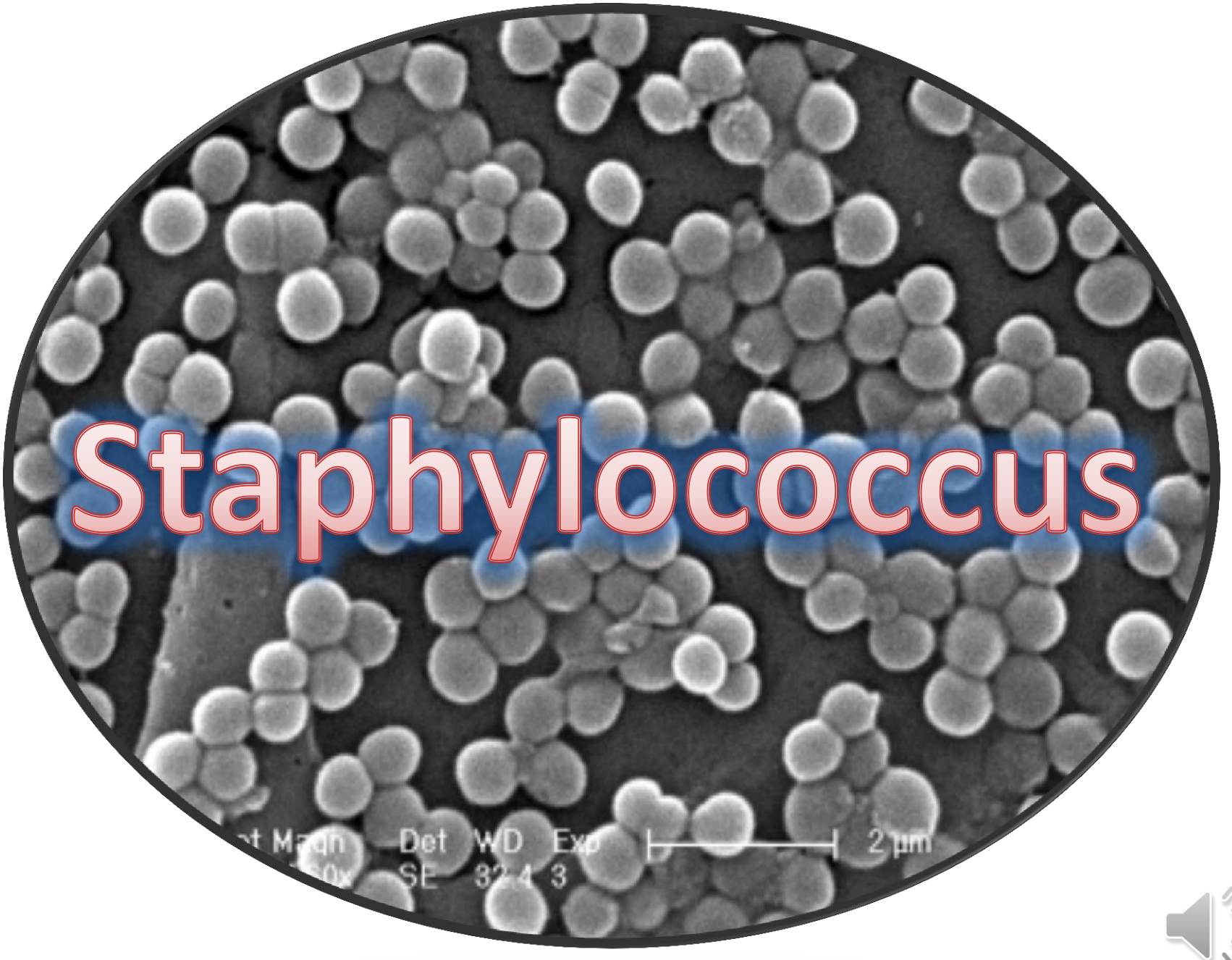
- **Present in motile strains except S.gallinarum**
- **- protein in nature**
- **- salmonella have 2 phases of flagellar antigens**

- **Phase 1 H** — **phase 2 H**
- **Identified by alpha-** — **20 H antigens are**
- **betical a,b,c,d ….** — **Signified by 2 numbers**
- — **1,2 – 1,3 etc**
- — **or 2 -3 letters as I,W , enx**
- — **enz ……**

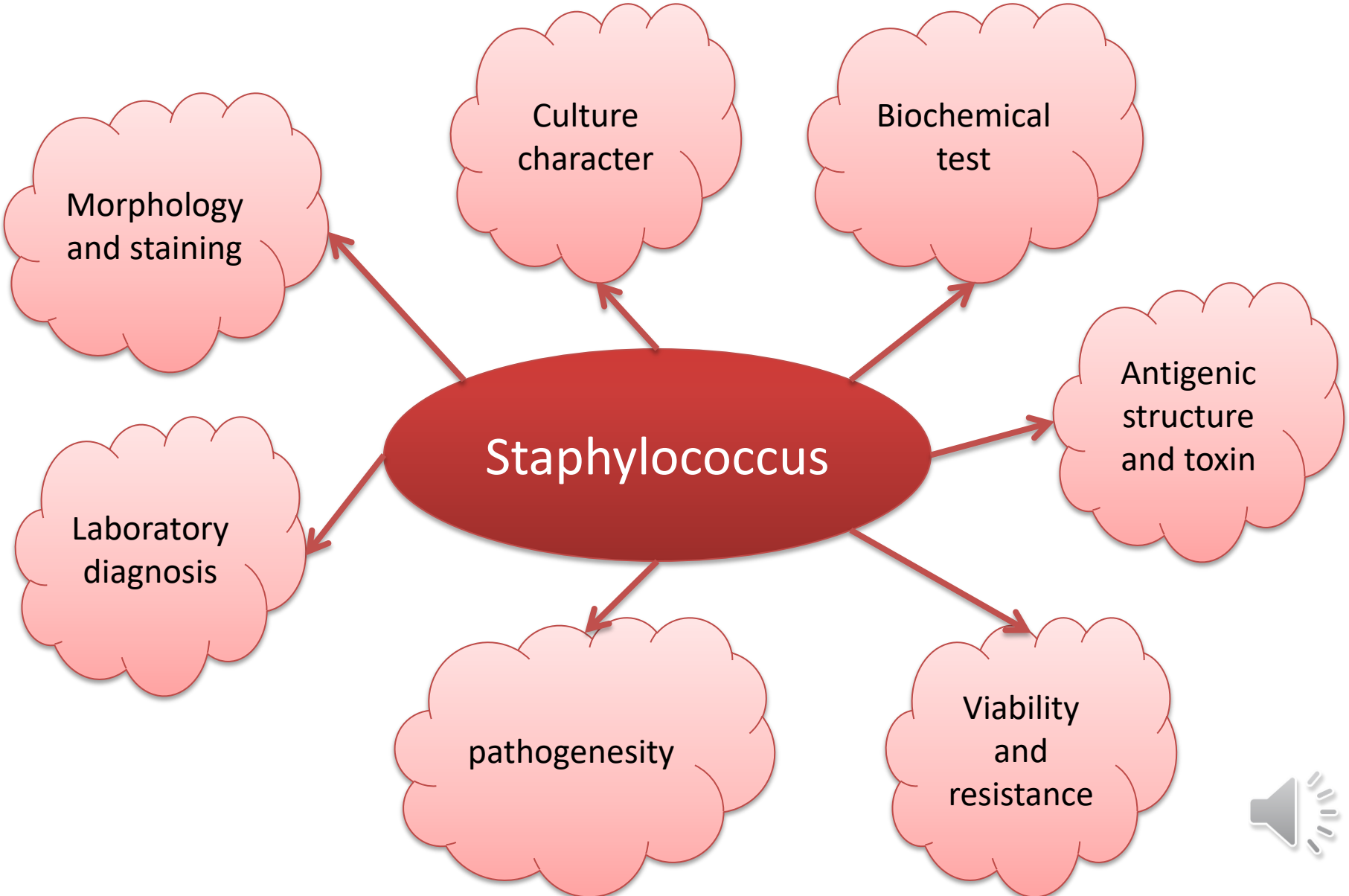
- **Virulent antigen (Vi – antigen)**
- **Present in freshly isolates of S.typhi**
- **Vi – antigen lost by subculturing**

Examples of salmonella serotypes

	S. paratyphi A	S. paratyphi B
group :	A	B
O-antigen :	1,2,12	1,4,5,12
H-antigen		
phase 1 :	a	b
phase 2 :	-	1,2
	monophasic	Diphasic

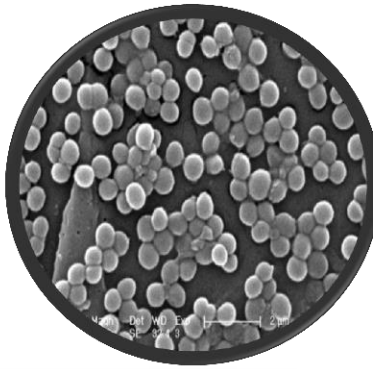

Staphylococcus
Det WD Exp
SE
2 µm

What must I know in microbe?



Morphological character and staining

Gram reaction	gram-positive cocci violet in color
shape	spherical or ovoid cells arranged in grapes like cluster
motility	non motile
capsule	non capsulated
Spore formation	non sporulated



culture character

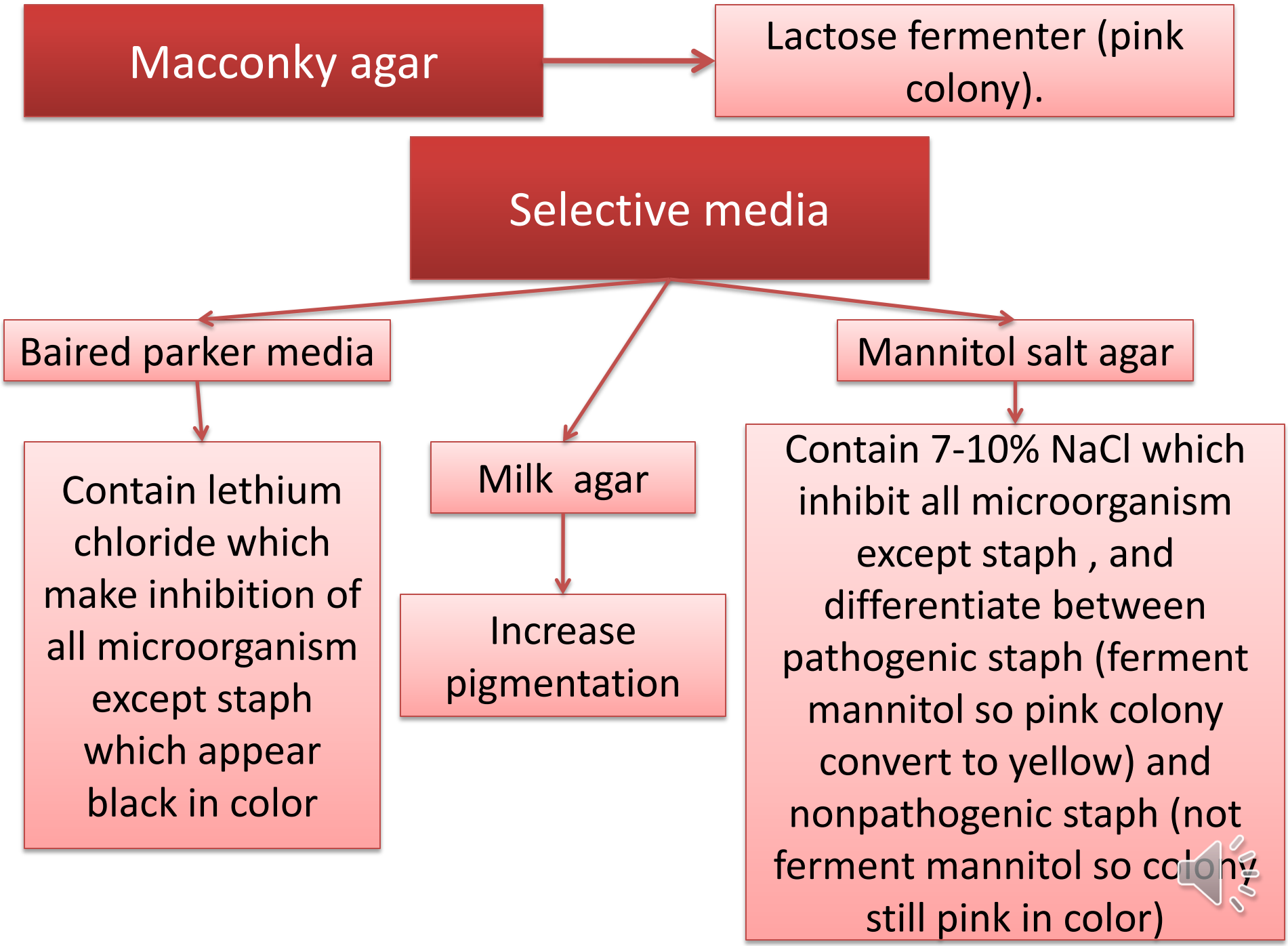
Grow of microbe on : media need to

oxygen	aerobic and facultative anaerobic
Temperature	37 celsius degree
PH	7.2
Incubation period	After 24 hours

culture character

media

Blood agar	are round, convex smooth, golden or white or lemon in color, with zone of complete haemollysis
Nutrient broth	uniform turbidity is present with powdery sediment and no pigments.
Nutrient agar	Small smooth endopigmented colony (colony only pigmented)


Macconky agar
Lactose fermenter (pink colony).
Selective media
Baired parker media
Mannitol salt agar
Contain lethium chloride which make inhibition of all microorganism except staph which appear black in color
Milk agar
Increase pigmentation
Contain 7-10% NaCl which inhibit all microorganism except staph , and differentiate between pathogenic staph (ferment mannitol so pink colony convert to yellow) and nonpathogenic staph (not ferment mannitol so colony still pink in color)

Biochemical test

Sugar fermentation test	as lactose, glucose, mannitol giving acid only
indol	-Ve
Methyle red(M.R)	+Ve
Vogus proskauer(V.P)	+Ve
Coagulase test	with pathogenic strain +Ve (clotting serum)
Catalase test	+Ve

Antigenic character and Toxin

Staph.aureus produces the following exo-toxins and enzymes:

1-coagulase enzyme:

Coagulated citrated plasma and it is used to differentiate between pathogenic and non-pathogenic staphylococcus strains, as in this picture which show postitive coagulase at the left (*S.aureus*) and negative at the right(non pathogenic strain)

2-leucocidin :

It is a substance which destroys leucocytes, haematoblasts of the bone marrow.

3-fibrinolysin: dissolve blood clot.

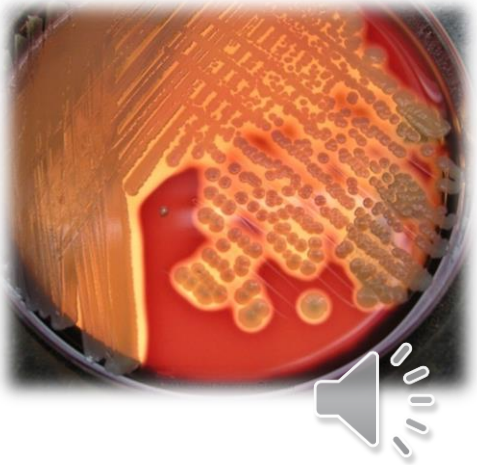
4-**hyalourinidase enzyme**: (spreading factor) it breaks down the hyalouronic acid.

5-haemolysins:

A substance which causes haemolysis to R.B.Cs and this organism has 3 types of haemolysins:

.

α-haemolysin:	- β-Haemolysin:	γ-Haemolysin:
causing haemolysis of rabbbit R.B.Cs ,sheep R.B.Cs.	- causing haemolysis of sheep R.B.cs ,weak on rabbit	causing haemolysis of rabbit R.B.Cs ,sheep R.B.Cs,human R.B.Cs



6-Enterotoxin:

It causes food poisoning in man. This toxin did not destroyed by 100 c for 30 minute while N/100 HCL destroys the enterotoxin.

7-lethal toxin:

It causes death in rabbits, cats, guinea pigs and mouse.

8-Dermonecrotoxin:

It causes necrosis of skin; it has been demonstrated experimentally in the rabbit and occurs in natural infection in the pastular dermatitis in dogs

Viability and resistance

-it is the most resistant of cocci.
-killed by heating at (60-80 C) for 30 minutes.
-killed by gentian violet at 1/25,000 after 30 sec.
-killed by 10%formaldhyde in 10 minutes
-it is very sensitive to the antibiotics such as penicillin, streptomycin and neomycin

•there are penicillin resistant strains (MRSA) (methicillin resistant staphylococcus aureus)

The penicillin normally affected on staphylococcus by B-lactam which affected on ability of bacteria to produce mucopeptide which is the main component of cell wall (essential structure) while resistant strain have ability to produce B-lactamase enzyme which make destruct of penicillin B-lactam so no effect of penicillin antibiotic with this modefied strain (resistant strain)

pathogenesity

Staphylococcus aureus usually associated with the following:

-Suppurative wound infection in man and animals.

- Mastitis in animals.

- pastular dermatitis in dogs.

-Food poisoning in man.

-Botryomycosis in horse.

Laboratory diagnosis

pus Sample:

Direct smears: from pus stained with gram stain to demonstrate gram positive cocci arranged in grape like clusters.

Isolation of the organism on specific media as mannitol salt agar.

Detection of the enterotoxin in case of food poisoning.

Thank you for attention

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

IMMUNOLOGY

INTRODUCTION

SECTION NUMBER ONE

1-OUR ENVIRONMENT CONTAINS AGREAT VARIETY OF INFECTIOUS MICROBES WHICH CAN CAUSE DISEASE

2-MOST INFECTIONS IN NORMAL INDIVIDUALS ARE SHORT LIVED AND LEAVE PERMENANT DEMAGE

3-THE DAMAGES ARE DUE TO THE IMMUNE SYSTEM WHICH COMBATS INFECTIOUS AGENTS 4-SINCE MICRO-ORGANISMS COMES IN DIFFERENT FORMS AWIDE VARIETY OF IMMUNE RESPONSES ARE REQUIRED TO DEAL WITH EACH TYPE OF INFECTIONS .

5-THE SITE OF INFECTION AND THE TYPE OF PATHOGEN LARGELY DETERMINES WHICH IMMUNE RESPONSE WILL BE EFFECTIVE

6-THE MOST IMPORTANT DISTINCTION IS BETWEEN THOSE WHICH INVADE THE HOST CELLS.

(وضوح – تباين – علامة فارقة- فرق-جلاء –إختلاف – طبقة- منزلة)

7-ANY IMMUNE RESPONSE INVOLVES FIRSTLY ,RECOGNITION OF THE PATHOGEN OR OTHER FOREIGN MATERIALS AND SECOUNDLY ,MOUNTING OF AREACTION AGAINST IT TO ELIMINATE IT .

THE TERM IMMUNITY (LAT.IMMUNIS= FREED HOMAGE , SAVE FROM SOMETHING) USUALLY MEANS RESISTANCE OF THE BODY TO PATHOGENS ,THEIR TOXINS OR TO OTHER KINDS OF THE FOREIGN SUBSTANCES.

TYPES OF IMMUNITY

MODERN CLASSIFICATION SUBDIVIDES IMMUNITY INTO TWO TYPES

1-INNATE (NON-SPECIFIC) IMMUNITY 2-ACQUIRED (ADAPTIVE) IMMUNITY

THE INNATE IMMUNITY

IS CHARACTERIZED BY BEING :

1-NON-SPECIFIC

2-DOES NOT DEPEND UPON PREVIOUS EXPERIENCE WITH THE INFECTIVE ORGANISM

WHILE

THE ADAPTIVE IMMUNITY

IS DEPENDANT UPON FOUR PILLARS :

1-SPECIFICITY.

2-DIVERSITY.

3-SELF-NON-SELF RECOGNITION.

4-IMMUNOLOGIC MEMORY .

DIFFERENCE BETWEEN INNATE AND ADAPTIVE IMMUNITY

	INNATE	ADAPTIVE
CHARACTERISTICS		
1-SPECIFICITY	FOR STRUCTURES SHARED BY RELATED MICROBES	FOR ANTIGENS OF MICROBES AND FOR NON MICROBIAL ANTIGENS
2- diversity	limited ; germline-encoded	Very large ;receptors are produced somatic recombinations of gene segments
3- MEMORY	NON	yes
4-NON RE ACTIVITY TO SELF	yes	yes
Components		
5-CELLULAR AND CHEMICAL BARRIERS	SKIN MUCOSAL EPITHELIAL ANTIMICROBIAL CHEMICALS	LYMPHOCYTES IN EPITHELIAL ; ANTIBODIES SECRETED AT EPITHELIAL SURFACES
6-BLOOD PROTIENS	COMPLEMENT AND OTHERS	ANTIBODIES
7-CELLS	PHAGOCYTES (MACROPHAGES ,NEUTROPHILS) NATURAL KILLER CELLS	lymphocytes

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

SECTION NUMBER 2

DOCTOR

MOHAMMED MAHMOUD

MOHAMMED YOUSEF WEHABA

TYPES OF IMMUNITY

INNATE (NON-SPECIFIC) IMMUNITY

THE HEALTHY INDIVIDUAL IS ABLE TO PROTECT HIMSELF FROM POTENTIALLY HARMFUL MICROORGANISMS IN THE ENVIRONMENT BY A VARIETY OF VERY EFFECTIVE MECHANISMS, PRESENT FROM BIRTH. THE MAIN DETERMINANTS OF INNATE IMMUNITY SEEM TO BE GENETICALLY CONTROLLED, VARYING WIDELY WITH SPECIES AND STRAIN AND TO LESSER EXTEND BETWEEN INDIVIDUALS. OTHER FACTORS LIKE AGE, SEX AND HORMONES BALANCE MAY PLAY A ROLE.

INNATE IMMUNITY IS DEPENDENTS ON FOUR DEFENSIVE BARRIERS ON FOUR DEFENSIVE BARRIERS: ANATOMIC, PHAGOCYTIC, ENDOCYTIC AND INFLAMMATORY.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

SECTION NUMBER 3

DOCTOR

MOHAMMED MAHMOUD

MOHAMMED YOUSEF WEHABA

INNATE IMMUNITY (NON-SPECIFIC):

I WILL COVER THOSE EVENTS BRIEFLY AND IN DETAILS OF THE FOLLOWING FOUR DEFENCIVE BARRIERS :

1-ANTOMIC 2-PHYSIOLOGIC 3-ENDOCYTIC 4-INFLAMMATORY.

1-ANATOMICAL BARRIERS INCLUDES :

A- INTACT SKIN AND SURFACE SECRETIONS.

B-MUCOUS MEMBRANES .

C-GASTEROINTESTINAL TRACT.

D- RESPIRATORY TRACT.

E- GENITAL SYSTEM .

F- EYES .

2-PHYSIOLOGICAL BARRIERS: INCLUDES :

A-PH .

B-TEMPRATURE.

C-OXYGEN TENSION .

D-VARIOUS SOLUBLE FACTORS.

N.B SOULBLE FACTORS:

WITHIN THE BODY FLUIDS MAJORLY AND INCLUDING THE SECRETED MUCOUS,IS AN ARRAY OF SOLUBLE SUBSTANCES WHICH HAVE PROTECTIVE FUNCTIONS BY:

(1)-INHIBITING THE GROWTH OF MICROORGANISMS,

(11)-OR NEUTRALIZING ENZYMES ON WHICH THE PATHOGENS DEPENDS.

Soluble factors comprises those :

(1)-transferin .

(11)-enzymes inhibitors .

(111)-lysins .

(V)-lysozyme .

(1V)-interferon .

(V1)-serum protiens .

(V11)- complement .

3-ENDOCYIC AND PHAGOCYTIC BARRIERS :

1-MEANS INGESTION OF EXTRACELLLULAR AMTERIALS.

2-NATURALLY PRESENT IN UNICELLULAR ORGANISMS.

3-CARRIED VIRTUALLY BY ALL CELLS.

4-PROFESSIONALLY BY BLOOD MONOCYTES,NEUTROPHILS, TISSUE MACROPHAGES.

5-STRATEGICALLY PLACED WHERE THEY ENCOUNTER INFECTIOUS AGENTS AS THE KUPFFER CELLS OF THE LIVER,SYNOVIAL A CELLS LINES THE SYNOVIAL CAVITY.

6-MONOCYTES DEVELOPES INTO TISSUE MACROPHAGES.

7-THIS PROCESS ARE VERY EFFECTIVE AT PRESENTING ANTIGENS TO T-LYMPHOCYTES.

7-TISSUE MACROPHAGES ARE:

1-BRAIN MICROGLIAL CELLS .

2-ALVEOLAR MCROPHAGES.

3-KIDNEY MESANGIAL MACROPHAGS.

4-OTHERS BLOOD CELLS ON PHAGOCYTOSIS:

A-Neutorphils B-Eosinophils C-Basophils.

4-BARRIERS CREATED BY THE INFLAMMATORY RESPONSE :

DEFINITION: ACOMPLEX SEQUENCE OF EVENTS COLLECTIVELY CAUSED BY TISSUE DAMAGES DISSEMINATED BY :

1-WOUNDS 2-APATHOGENIC MICRO-ORGANISMS .

1-MANY YEARS AGO VIA THE REMEDEYERS DESCRIBED THE CLASSIC FEATURES OF INFLAMMATIONS AS FOLLOWS:

1-REDNESS2-SWELLING3-HEAT4-PAIN5-LOSS OF FUNCTION.

2-THE CARDINAL SIGNS OF INFLAMMATION REFLECT THREE MAJOR EVENTS THAT OCCUR IN AN INFLAMMATORY RESPONSE :

1)-INCREASE BLOOD FLOW 2)-INCREASED CAPILLARY PERMEABILITY 3)-INFLUX OF PHAGOCYTIC CELLS.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

SECTION NUMBER 4

DOCTOR

MOHAMMED MAHMOUD

MOHAMMED YOUSEF WEHABA

ANTIGENS

ANTIGENS ((GR . ANTI MEANS AGAINST AND GEN MEANS TO FORM))

1-DEFINITION :-

A-ARE MACROMOLECULES SUBSTANCES OF VARIOUS CHEMICAL TYPES

B-WHICH INTRODUCED IN ANIMALS STIMULATES THE FORMATIONS OF :

1- ANTIBODIES 2-LYMPHOKINES

3-REGULATOR AND EFFECTOR T-LYMPHOCYTES

THOSE MEANS INTEGRATED IMMUNE RESPONSE .

CONDITIONS OF THE ANTIGENICITY :

1-FOREIGNNESS. 2-HIGH MOLECULAR WEIGHT .

3-PHYSICAL STATE OF ANTIGEN INCLUDE A-SOLUBILITY B-ELECTRIC CHARGE 4-CHEMICAL PROPERITIES DETAILED AS FOLLOWS:

A-POLYPEPTIDES B-PROTIENS C- POLYSACCHARIDES.

D-GLYCOPRPTIENS E-PEPTIDOGLYCANS F-NUCLOPROTIENS G-LIPOPROTIENS H-LIPOPOLYSACCHARIDES 5-DIGESTIBILTY.

6-BIOLOGICAL PROPERITIES.

N.B: 1- IMMUNOGENS AND HAPTENS.
2-ANTIGENIC DETERMINANTS.

CATEGORIES OF ANTIGENS :

1-HETEROGENIC ANTIGENS (HETEROPHIL ANTIGENS).
2-SPECIES SPECIFIC ANTIGENS .
3-ISO-ANTIGENS.
4-ORGAN SPECIFIC ANTIGENS.

N.B: 1-BACTERIAL ANTIGENS INCLUDES :
A-SOMATIC ANTIGENS B-FLAGELLAR ANTIGENS C-SURFACE ANTIGENS
D- VIRULENT ANTIGENS E-SPORES ANTIGENS

IMMUNITY

LECTURES WRITTEN AND PRESENTED BY

SECTION NUMBER 5

DOCTOR

MOHAMMED MAHMOUD

MOHAMMED YOUSEF WEHABA

IMMUNE RESPONSE

IMMUNE RESPONSE TAKES PLACE IN TWO FORMS:
1-HUMORAL IMMUNE RESPONSE 2-CELL-MEDIATED IMMUNE RESPONSE 1-HUMORAL IMMUNE RESPONSE:
THE CHARASTERISTIC OF THS FORM OF IMMUNITY IS (WHAT) =THE APPEARANCE IN BLOOD OF GLOBULINS KNOWN AS IMMUNOGLOBULINS OR ANTI bodies .these combine specifically with the antigen which stimulate their production and can lead to some remarkable consequences such as :
1-agglutination 2-neutralization of toxin
It is dependant on b-lymphocytes

Cell-mediated immune response :

1-occur particularly in infections by agents which enter the cells

2-agents includes :a-viruses b- tubercle bacilli c- brucella species

3-it can develop after skin contact with certain chemical substances

4-the sequence of steps leading to this form of immunity is essentially not different from that leading to antibodies response , but the response is initiated in different areas of the spleen and lymph nodes.

In simple diagrammatic steps of illustrating immunity :

Humoral immunity :

Extra cellular microbes leading to the responding via B-lymphocytes which by the effector mechanisms leading to the secretions of antibodies via serum as away of the transferring methods so enabling the antibodies to perform their functions which leads to block infections and eliminating the extra cellular microbes .

Cell-mediated immunity

The helper T-lymphocytes via their structures and abilities are leading to activations of the macrophages to kill phagocytosed microbes and the cyto-toxic lymphocytes kill infected cells and eliminating reservoirs of infections.

IMMUNITY

LECTURES WRITTEN AND PRESENTED BY

SECTION NUMBER 6

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

TISSUES INVOLVED IN IMMUNE RESPONSE

Tissues involved in immune response

1-the lymphoid tissues concerned with the immune response are located in primary organs as in (bone marrow – bursa of fabricius – thymus glands)

2-the lymphoid tissues concerned with the immune response are located in secondary organs as in (lymph nodes – spleen-tissues associates with the gut)

3-the lung and the liver may play a lesser roles .

4-the primary organs the central organs are responsible for the development and control of : 1- peripheral lymphoid tissues 2- the secondary lymphoid organs .

Nota benna : lymph nodes and spleen can be regarded as a complex organization of three types of cells involved in initiation of immune response as follow :

1-lymphocytes 2-plasma cells

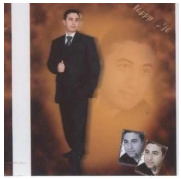
3-phagocytes of reticulo – endothelial system

What are lymphoid tissues and their functions :

1~consist of lymph nodes and spleen .

2~the spleen and lymph nodes forms the lymphocytes and plasma cells responsible for humeral antibodies responses.

3~ there are some areas of lymphoid tissues present in these organs under the influence of thymus called (thymus dependent areas) responsible for cell mediated immunity.



JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
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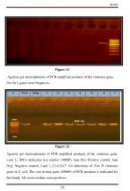

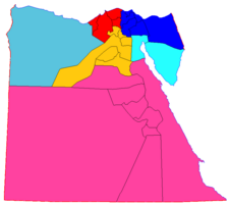

أعوذ بالله من الشيطان الرجيم

بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

(ذلك من أنباء القرى نقصه عليك منها قائم وحصيد)

الأية ١٠٠ من سورة هود

بحث الدكتوراة وعليه ١٥ درجة فى التقييم العام ويتبع قسم الباثولوجيا الإكلينيكية للحصول على درجة الدكتوراة من جامعة بنها كلية الطب البيطرى بمدينة مشتهر محافظة القليوبية تم عرضه وإختياره وتنزيله من على شبكة المعلومات الدولية و ذلك لدراسة إمكانياته وتقيمه من ضمن الأجهزة الدقيقة فى تشخيص الأمراض المميتة الوبائية منها والوافدة وكيفية علاجها والحد من أخطارها.مقدمة من باحث مساعد

محمدمحمودمحمديوسف وهيبه .ماجستير البكتيريا والمناعة والفطريات بمعهد بحوث الصحة الحيوانية فرع طنطا التابع لمركز البحوث الزراعية التابع لوزارة الزراعة بجمهورية مصر العربية. للدراسة من قبل قسم الباثولوجيا الإكلينيكية .كلية الطب البيطرى بمشتهر.جامعة بنها العريقة.

رقم الجلوس الإختبارى : ٣٠١٤ المقرر : ٨١٤٢ .

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

لديكم كل محاضراتى ورسائلى على موقع التعليم الإلكترونى

باحث مساعد محمدمحمودمحمديوسف وهيبه

رقم الجلوس ٢٠١٤/٣جامعة بنها ودليل معلوماتكم

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Applications of Flow Cytometry to Clinical Microbiology

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We dedicate this review to Luis Carrasco.

INTRODUCTION

Microbiology in general and clinical microbiology in partic- ular have witnessed important changes during the last few years (82). An issue for microbiology laboratories compared with other clinical laboratories is the relative slowness of de- finitive reports. Traditional methods of bacteriology and my- cology require the isolation of the organism prior to identifi- cation and other possible testing. In most cases, culture results are available in 48 to 72 h. Virus isolation in cell cultures and detection of specific antibodies have been widely used for the diagnosis of viral infections (181). These methods are sensitive and specific, but, again, the time required for virus isolation is quite long and is governed by viral replication times. Addition- ally, serological assays on serum from infected patients are more useful for determining chronic than acute infections. Life-threatening infections require prompt antimicrobial ther- apy and therefore need rapid and accurate diagnostic tests. Procedures which do not require culture and which detect the presence of antigens or the host's specific immune response have shortened the diagnostic time. More recently, the emer- gence of molecular biology techniques, particularly those based on nucleic acid probes combined with amplification techniques, has provided speediness and specificity to micro- biological diagnosis (139). These techniques have led to a revolutionary change in many of the traditional routines used

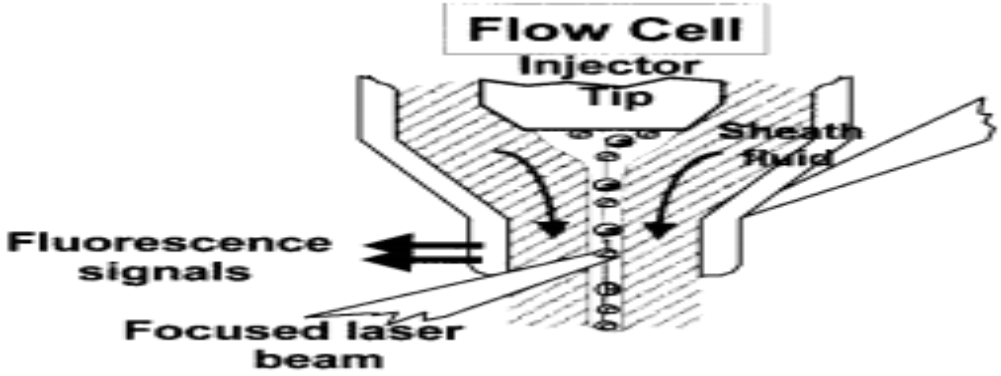


FIG. 1. Light-scattering and fluorescence signal production at the flow cell analysis point of the flow cytometer. From Purdue Cytometry CD-ROM vol. 1 (adapted with permission of the publisher).

in clinical microbiology laboratories. Results are offered quickly, the diagnosis of emerging infections has become easier, and unculturable pathogens have been identified (109). On the other hand, the current organization of clinical microbiology laboratories is now subject to automation and competition, both overshadowed by increasing costs (282, 339). Increased use of automation in clinical microbiology laboratories is best exemplified by systems used for detecting bacteremia, screening of urinary tract infections, antimicrobial susceptibility testing, and antibody detection. To obtain better sensitivity and speed, manufacturers continuously modify all these systems. Nevertheless, the equipment needed for all these approaches is different, and therefore the initial costs, both in equipment and materials, are high.

Flow cytometry (FCM) could be successfully applied to mostof these situations. In bacteremia and bacteriuria, FCM would not only rapidly detect organisms responsible for the infection but would also initially identify the type of microorganism on the basis of its cytometric characteristics. Although FCM offers a broad range of potential applications for susceptibility test- ing, a major contribution would be in testing for slow-growing microorganisms, such as mycobacteria and fungi (108, 163, 262). Results are obtained rapidly, frequently in less than 4 h; when appropriately combined with the classical techniques, FCM may offer susceptibility results even before the microor- ganism has been identified. The most outstanding contribution offered by FCM is the detection of mixed populations, which may respond to antimicrobial agents in different ways (331).

This technique could also be applied to study the immune response in patients, detect specific antibodies (27, 133), and monitor clinical status after antimicrobial treatments (58, 244). Moreover, when properly applied, FCM can be adjusted to use defined parameters that avoid subjectivity and aid the clinical microbiologist in the interpretation of specific results, partic- ularly in the field of rapid diagnosis.

TECHNICAL BASIS OF FLOW CYTOMETRY

FCM is an analytical method that allows the rapid measure- ment oflight scattered and fluorescenceemission produced by suitably illuminated cells. The cells, or particles, are suspended in liquid and produce signals when they pass individually through a beam of light (Fig. 1). Since measurements of each particle or cell are made separately, the results represent cu- mulative individual cytometric characteristics. An important analytical feature of flow cytometers is their ability to measure multiple cellular parameters (analytical flow cytometers). Some flow cytometers are able to physically separate cell sub- sets (sorting) based on their cytometric characteristics (cell sorters) (Fig. 2). The scattered light (intrinsic parameters) and fluorescence emissions of each particle are collected by detec- tors and sent to a computer, where the distribution of the population with respect to the different parameters is repre- sented. Scattered light collected in the same direction as the incident light is related to cell size, and scattered light collected at an angle of 90° gives an idea of the particle complexity. This parameter is related to cell surface roughness and the number of organelles present in the cell. Size and complexity are con- sidered intrinsic parameters since they can be obtained without having to stain the sample.

To obtain additional information, samples can be stained using different fluorochromes. Fluoro- chromes can be classified according to their mechanism of action (127): those whose fluorescence increases with binding to specific cell compounds such as proteins (fluorescein iso- thiocyanate [FITC]), nucleic acids (propidium iodide [PI]), and lipids (Nile Red); those whose fluorescence depends on cellular physiological parameters (pH, membrane potential, etc.); and those whose fluorescence depends on enzymatic activity (fluorogenic substrates) such as esterases, peroxidases, and peptidases (Table 1). Fluorochromes can also be conju- gated to antibodies or nucleotide probes to directly detect microbial antigens or DNA and RNA sequences.

1-SCHEMEمخطط

2-OPTIC قديح بصرى

3-DICHROIC مزدوج اللون

4-MIRRORS مرايا – دولاب مرايا – مرايا المسح

5-BANDPASS تمرير الحزم الضوئيات –تمرير النطاق –ممر الموجة – إجتياز نطاق التردد

6-FILTERS تقتية مرشحات التمرير النطاقية –مرشح امرار حزمة – مصفاة ممر النطاق-مرشح إمرار الحزمة-المرشح القابل – مرشح تمرير نطاقى -

7-ILLUMINATIONS إنارة-إضاءة زينية – الإشراق عندالصوفية-إضاءة الإستصباح-التذهيب-إضاءة-إنارة-نمنمة-نور-إضاءة سطحية وإضاءة مباشرة –مضواء-إضاءة الساعة-الإضاءات داخلية وأرضية

8-LASER أشعة الليزر- لا زر –ميزربصرى-مضخم الأمواج الدقيقة بالإبتعاث الإشعاعى المستثار بالطاقة الضوئية

9-SYSTEMS

10-PARAMETERS مؤشرات وإحداثيات مقاييس وأبعاد متغيرات ومعالم

11-GRANUALRITY حبوبية و التغلظ الوظيفى

12-PHOTODIODE ليزر أشباه المواصلات تساوى دايود الليزر فهم شكل من أشكال التقنية .

13-PMT TUBE photomultiplier tube أنبوب مضخم للضوء أنبوب مضاعف ضوئى صمام مضاعف ضوئى إلكترونى

14-SORTING CAPACITY قدرة الفرز و القدرة التصنيفية والإخراج والتنحية يعاشر بالفلوسيتوميترى

15-PURUDE

16-PURUDE CYTOMETERY CD-ROM

LASER MEANS LIGHT AMPLIFICATIONS BY STIMULATED EMISSIONS OF RADIATIONS

تضخيم الضوء بالإنبعاث المحفز للإشعاع . هو جهاز ينبعث منه الضوء من خلال عملية تضخيم ضوئى تعتمد على الإنبعاث المستحث للإشعاع الكهرومغناطيسى . LASER هى الأحرف الأولى لمجموعة من الكلمات وهن كما سبق.

CD- ROM COMPACT DISC –READ-ONLY-MEMORY IS AN ADAPTATION OF THE CD THAT IS DESIGNED TO STORE COMPUTER DATA IN THE FORM OF TEXT AND GRAPHICS , AS WELL AS HI-FI STERO SOUND .

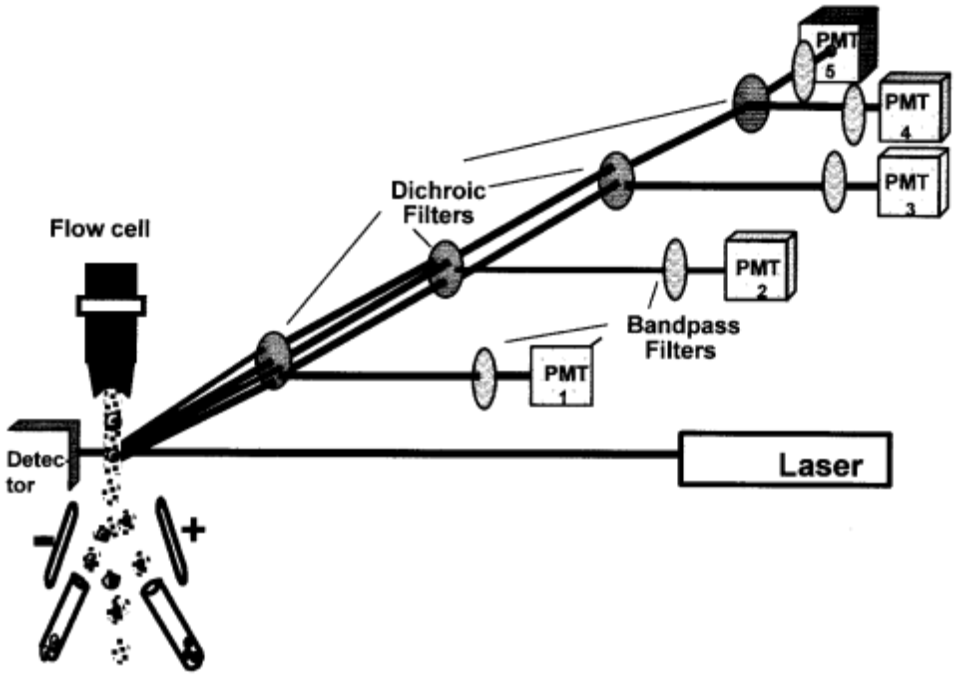


FIG. 2. Scheme of optic (dichroic mirrors and bandpass filters) and illumination (laser) systems of a flow cytometer with six parameters detected (size, granularity, and four fluorescences) by separate photomultiplier tubes (except size,which can be detected by photodiode or a PMT tube) and sorting capacity. From Purdue Cytometry CD-ROM vol. 1 (adapted with permission of the publisher).

A typical flow cytometer has several parts. (i) The hydraulic system produces the fluid stream, with a liquid sheath sur- rounding the cell suspension (hydrodynamic focusing). This sheath is responsible for the passage of the particles through the sensing point at a constant velocity. (ii) The illumination system consists of the light that produces the scatter signals and fluorescence emission when the particles pass through it. There are two types of flow cytometers, depending on the illumination source: those with a laser light source, and those with an arc lamp source. Each has it own advantages and disadvantages, but the main difference lies in their fields of application. Arc lamp cytometers are frequently used in mi- crobiological applications due to their better scatter resolution and versatility. In contrast, laser flow cytometers have wider applications in immunology and hematology because they ex- cite fluorochromes associated with cells. Studies comparing the two types of cytometers have concluded that the selection of one rather than the other depends mainly on the range of wavelengths required for the excitation of the selected fluores- cent stains (13, 161).

Our personal experience supports this opinion and work should aim at developing protocols accord- ing to the type of cytometer available.
(iii) The optic system focuses incident light on the crossing particles, recovers the scattered light and the fluorescence produced by the fluoro- chromes present in the cells, and directs both to the appropri- ate photomultiplier tubes (Fig. 2).
(iv) The electronic system transforms the incident light from fluorescence and light scat- tered into electric pulses (analogic).

The magnitudes of these pulses are distributed electronically into channels, permitting the display of histograms of the number of cells plotted against the channel numbers (digital) (68). If the instrument has the capacity to do so, it also controls the cell-sorting process. Flu- orescence-activated cell sorting refers to the ability to select a subpopulation from the whole population, following cytomet- ric classification, and to physically separate this particular pop- ulation. To do this, the machine produces a uniform stream of droplets; a particular droplet containing a cell can be charged, permitting selection of the droplet when it passes through an electrical field produced by deflection plates (Fig. 2). In this way, two populations can be sorted at the same time (positively and negatively charged droplets). A new high-speed sorter machine has been developed with the possibility of sorting four populations at the same time (MoFlo; Cytomation, Freiburg, Germany).

(v) The data analysis system consists of software that allows the analysis of the huge amount of information produced by multiparameter data acquisition. The analytical software permits the study and independent analysis of a par- ticular subpopulation. Besides all the statistical information, the data can be represented in several different ways: mono- parametric histograms, biparametric histograms, and three- dimensional representations (Fig. 3). There is a growing mar- ket of commercial FCM software. Free software can also be downloaded from the Internet, where it is possible to find information about all the fields related to FCM (cytometry network sites, http://nucleus.immunol.washington.edu/ISAC /network_sites.html; JCSMR flow cytometry software, http:// jcsmr.anu.edu.au/facslab/facs.html; ISAC WWW home page, http://www10.uniovi.es/ISAC.html).

FLOW CYTOMETRY AND MICROBIOLOGY, A LONG TIME TOGETHER

From the beginning of FCM (68), the ancestor of modern flow cytometers has been identified with an aerosol particle counter designed to analyze mine dust (124). This apparatus was used in World War II by the U.S. Army in experiments for the detection of bacteria and spores. Gucker et al. (124) reported that the instrument could be used with biological samples (bacteria), as well as particles in air suspension or aerosols. Thus, FCM with an application to microbiology originated many years before the use of flow cytometry as a tool for studying mammalian cells. The original device incorporated a sheath of filtered air to limit the air sample stream to the central portion of the flow chamber. The detector used was a then recently developed device called a photomultiplier tube. Particle counters based on the Coulter orifice principle, in which the difference in electrical conductivity between the cells and the medium in which they are suspended is measured by the change in electrical impedance produced as they pass through an orifice, were later developed.

These instruments were widely applied in hematology studies. However, the first real flow cytometer was built by Kamentsky et al. (154), using spectophotometric techniques to detect and measure nucleic acids and light scattering of unstained cervical cells in a flow stream. At the same time, Fulwyler, working at the Los Alamos Scientific Laboratory, described the first flow cytometer with sorting capability (104). This machine worked by measuring cell volumes obtained by the Coulter orifice principle. Fulwyler adapted the inkjet printer principle, using electrostatic deflec- tion of charged droplets, as a cell-sorting mechanism. In fact, sorting capability was introduced to demonstrate the accuracy of the signals obtained by the machine and to ascribe a given distribution of cell volume detected by an electronic signal to a specific cell type.

During the 1970s, applications of FCM to research into mammalian cells advanced rapidly, but at that time few instruments were developed for microbiological studies. The subsequent applications to microbiology ofFCM techniques that were initially developed to study mammalian cells were due to optical improvements in flow cytometers and newly developed fluorochromes. The development of an arc lamp-based instrument by Steen's group in 1979 (301, 303) allowed the use of FCM for basic research on bacteria. Because of the design of the flow chamber and the use of photomultiplier tubes for detecting scattered light, this instrument was ideal for studying microorganisms (7, 37).

The promising tool described by Boye and Steen in 1983 became a "potent illuminating light" in the 1990s (38), as was stated in the book edited by David Lloyd, *Flow Cytometry in Microbiology* (186a), from which most microbiological cytometrists have learned their trade.

TABLE 1. Some of the fluorescent molecules used to study microorganisms by flow cytometry

Dye	Excitation wavelength (λ_{max}) (nm)	Emission wavelength (λ_{max}) (nm)	Ligand or substrate	Applications
TOTO-3	642	660	DNA, RNA	DNA quantification, cell cycle studies
SYTOX Green	504	525	DNA, RNA	Viability, DNA quantification
PI	536	625	DNA, RNA	Viability, DNA quantification, cell cycle studies
Ethidium bromide	510	595	DNA, RNA	DNA quantification, cell cycle studies
Hoechst 33258/33342	340	450	DNA (GC pairs)	Cell cycle studies
SYTO 13	488	509	DNA, RNA	Viability, DNA quantification, cell cycle studies
Mithramycin	425	550	DNA	Cell cycle studies
Pyronine Y	497	563	RNA	RNA quantification
FITC	495	525	Protein	Microbe detection
Texas Red (sulforhodamine isothiocyanate)	580	620	Protein	Microbe detection
Oregon Green isothiocyanate	496	526	Protein	Microbe detection
Indo-1	340	398–485	Ca^{2+}	Ca^{2+} mobilization
Fura-2	340	549	Ca^{2+}	Ca^{2+} mobilization
Fluor-3	469	545	Ca^{2+}	Ca^{2+} mobilization

BCECF	460–510	520–610	pH	Metabolic variations
SNARF-1	510	587–635	pH	Metabolic variations
$DIOC_6(3)$	484	501	Membrane potential	Antibiotic susceptibility, metabolic variations
Oxonol [$DiBAC_4(3)$]	488	525	Membrane potential	Antibiotic susceptibility, metabolic variations
Rhodamine 123	507	529	Membrane potential (mitochondria)	Antibiotic susceptibility, metabolic variations
Fun-1	508	525–590	Yeast vacuolar enzyme activity	Yeast metabolic state
Nile Red	490–550	540–630	Lipids	
Lectins	Depends on fluorochrome conjugated	Depends on fluorochrome conjugated	Membrane oligosaccharides	Cell wall composition, microbe detection
Fluorescently labeled oligonucleotides	Depends on fluorochrome conjugated	Depends on fluorochrome conjugated	Nucleotide sequences	Microbe identification
Calcofluor white	347	436	Chitin and other carbohydrate polymers	Fungal detection
Substrates linked to fluorochromes			Enzyme activities	Metabolic activity
Antibodies labeled with flurochormes			Antigens	Microbe detection

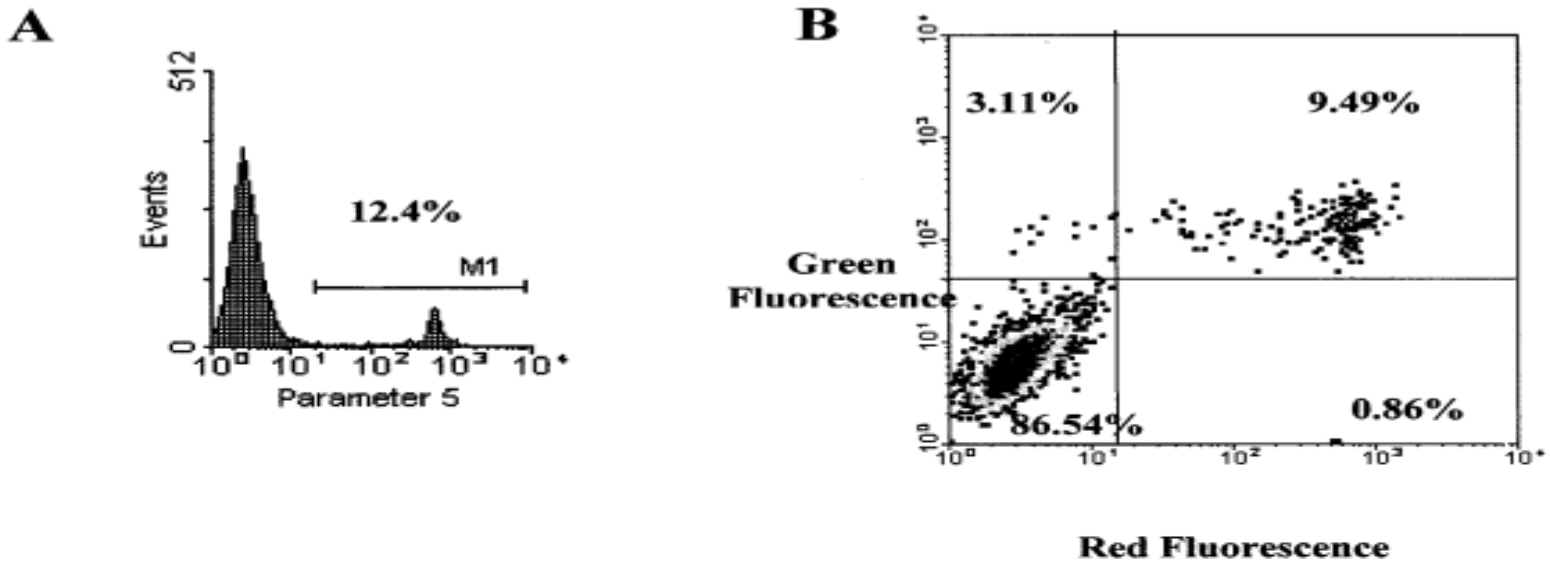

A
512
Events
0
12.4%
M1
10^0 10^1 10^2 10^3 10^4
Parameter 5
B
3.11%
9.49%
Green Fluorescence
86.54%
0.86%
Red Fluorescence
C
PI Fluorescence
Cell Size
D

FIG. 3. The data obtained from a flow cytometer can be displayed in several ways. The most common are the mono- and bi parametric histograms (Aand B), which usually include a statistical analysis of the results. (A) Mono parametric histogram showing the selected parameter on the *x* axis and the relative cell number on the *y* axis. (B) Bi parametric histogram showing cells distributed as a function of their signal intensity with respect to each parameter. Cells located in the upper left quadrant are positive for the parameter represented on the *y* axis, cells located in the upper right quadrant are positive for both parameters, cells located in the lower left quadrant are double negative, while cells in the lower right panel are positive for the parameters on the *x* axis. (C and D) Three-dimensional representations. The *z* axis can represent the relative number of cells (C) or a third parameter (D), such as scattered light on the *x* and *y* axes and fluorescence signals on the *z* axis.

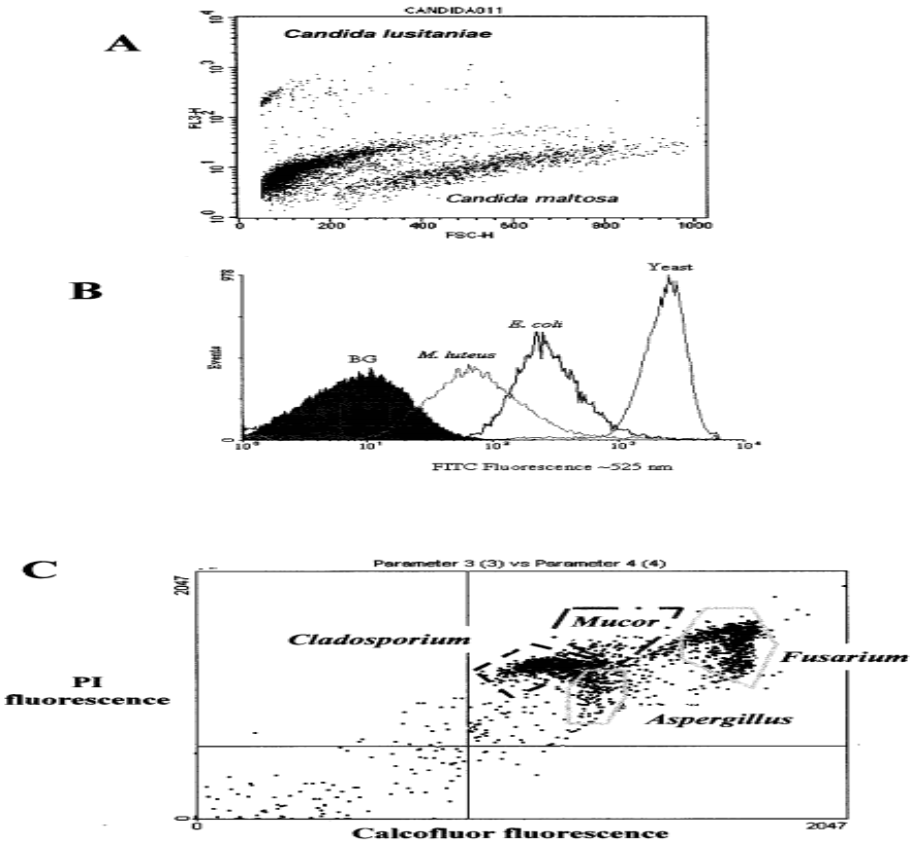
FCM in microbiology have significantly increased (9, 28, 103, 148, 291).

General Applications of Flow Cytometry to Microbiology

The applications of FCM to microbiology have been so widespread that discussion of all of them is beyond the scope of this review. For more information, see the excellent reviews by Davey and Kell (68), Porter et al. (263), and McSharry (211) and the "Bible" of flow cytometry by Howard Shapiro, *Practi- cal Flow Cytometry* (291). Below, we briefly describe some of the applications of FCM in the field of microbiology, focusing on present or future applications in clinical microbiology.

Earlier works by Steen had demonstrated the applicability of dual-parameter analysis to discriminate among different bac-teria in the same sample (301, 302, 306). One parameter was light scattered (size), and the other was either fluorescence emission from fluorochromes coupled to cellular components (protein and DNA) or autofluorescence (Fig. 4), or light scat- tered acquired from another angle (42, 151, 277, 293, 301, 302, 306, 316). However, the use of several fluorochromes for direct staining or through antibody or oligonucleotide conjugates plus size detection is the simplest way to visualize or identify microorganisms by FCM (6, 7, 10, 11, 257, 317, 332).

The simple and rapid assessment of the viability of a micro- organism is another important aspect of FCM. The effect of environmental stress or starvation on the membrane potential of bacteria has been studied by several groups using fluoro- chromes that distinguish among nonviable, viable, and dor- mant cells (155–157, 188; see references 68, 71, 78, 89, 135, 150, 200–203, 265, and 328 for reviews). Other authors have demonstrated the use of PI as a viability marker in yeasts (72), *Pneumocystis carinii* (177), and bacteria (89, 230, 264).


A
CANDIDA011
Candida lusitaniae
Candida maltosa
FL3-H
FSC-H
B
BG
M. luteus
E. coli
Yeast
FITC Fluorescence ~525 nm
C
Parameter 3 (3) vs Parameter 4 (4)
Cladosporium
Mucor
Fusarium
Aspergillus
PI fluorescence
Calcofluor fluorescence
2047

FCM has also been used in metabolic studies of microor- ganisms. This was first accomplished by Thorell (316), using autofluorescence due to NADPH and flavins as metabolic sta- tus markers. Other authors studied DNA, protein, peroxide production, and intracellular pH (3, 5, 39, 140, 324, 345). Re- cently developed fluorochromes and kits (Sytox Green and Live/Dead kits; Molecular Probes, Eugene, Oreg.) have been used for the FCM-based counting of live and dead bacteria and yeasts (176, 178, 311), simplifying staining protocols and mak- ing data interpretation easier. Other kits are available for de- tecting gram-positive and gram-negative bacteria or for study- ing yeast organelles (127). Recently, Mason et al. (204) described a method which enables the discrimination of gram- positive from gram-negative bacteria on the basis of the fluo- rescence emitted when the organisms are stained with two fluorochromes. These authors correctly predicted the Gram stain reaction of 45 strains of clinically relevant organisms, including several known to be gram variable. In addition, rep- resentative strains of gram-positive anaerobic organisms, which are normally decolorized during the traditional Gram stain procedure, were classified correctly by this method.

FCM also offers the possibility of studying gene expression using reporter genes in yeasts (56, 258, 297, 338) and bacteria (8, 55). The development of gene expression systems based on green fluorescent proteins facilitates this kind of study due to the simplicity of the technique (60, 77, 229, 320).

The sensitivity of FCM allowed Philips and Martin (255) to detect *Bacillus* spores (254). Using a similar approach, Griffiths et al. (121) and Challier et al. (49) were able to sort spores from *Dictyostelium discoideum* and *Enterocytozoon bieneusi*, re- spectively. These examples show the potential of FCM in the investigation of small microbes.

The interaction between pathogens and phagocytic cells has also been studied by FCM (22, 23). The development of fluo- rochromes to detect oxidative bursts due to phagocytosis (17, 251, 281) increased the number of studies with different mi- crobes such as *Borrelia burgdorferi* (17), *Staphylococcus* spp. (128, 196), *Escherichia coli* (70, 271), *Bordetella pertussis* (299), *Cryptococcus neoformans* (48), *Salmonella* (272), and yeasts (90, 96, 114).

FCM has been extensively used for studying virus-cell inter- actions (172, 180, 334). This topic was reviewed in depth by McSharry in 1994 (211). Modulation of the expression of cel- lular proteins due to viral infection has been studied by FCM for cytomegalovirus (CMV) (116), herpes simplex virus (HSV) (149), adenovirus (168), human immunodeficiency virus (HIV) (53), and hepatitis B virus (HBV) (346). Perturbation of the cell cycle and DNA replication in virus-infected cells have also been studied by FCM for papillomavirus (25), CMV (80) and human HIV-1 (273). This technique has been also used to study the effect of viral infection on intracellular Ca^{2+} levels ($[Ca^{2+}]i$) by Irurzun et al. (145) and by Miller et al. (221). FCM has also permitted the demonstration of apoptosis associated

with viral infection, including HSV (144, 146), Epstein-Barr virus (EBV) (4), influenza virus (267),measles virus (93), pap- illomavirus (340), and HIV-1 (129, 194) infections. Further- more, by means of biotinylated or directly FITC-labeled virus, interactions of EBV (142, 159, 182, 335), echovirus (206), ad- enovirus (217), influenza virus (228), simian virus 40 (SV40) (20), human T-cell leukemia virus type 1 (HTLV-1) (110), measles virus (226, 232), bovine herpesvirus (326), papilloma- virus (268), bunyaviruses (249), poliovirus (102), and HIV-1 (14, 308, 318) with their putative cell receptors have been described. These investigations show that FCM is able to pro- vide solutions to problems arising when working with micro- organisms.

APPLICATIONS OF FLOW CYTOMETRY TO CLINICAL MICROBIOLOGY

The isolation of microbes and their identification, the detec- tion of increased levels of antibodies to a particular pathogen in the course of an illness, and direct detection of microbial components (nucleic acids and proteins) in clinical samples obtained from different tissues or body fluids are the main tools for laboratory diagnoses of microbial infections. Effective antimicrobial therapies have indeed been developed because early treatment is crucial in many cases; therefore, rapid diag- nosis is essential in the fight against infection.

Direct Detection

Bacteria. Antibodies are currently changing the way in which we identify microbes, making it easier and faster. Their spec- ificity and the possibility of using fluorochrome-labeled anti- bodies to specific antigens render them one of the most pow- erful tools in the identification of pathogens. The main disadvantage of this method is still the limited availability of antibodies directed against particular microbes. Other advan- tages of using antibodies are that the cells do not need to be cultivable and that the method is simple and fast. In an early work from 1983, Groschel (122) explained somewhat prophet- ically the use of antibodies in clinical microbiology. FCM in conjunction with fluorescent antibodies has been used to de- tect surface antigens in *Haemophilus* (298), *Salmonella* (57, 207), *Mycobacterium* (238), *Brucella* (35), *Branhamella ca- tarrhalis* (29), *Mycoplasma fermentans* (50), *Pseudomonas aeruginosa* (134), *Bacteroides fragilis* (191, 239) and *Legionella* (143), among other microorganisms. These examples illustrate the sensitivity and specificity of using antibodies that allow the detection of particular cell types (of as few as 100 cells per ml in 30 min) in heterogeneous populations (57).

The first study detecting of microbes in blood by using FCM was done with ethidium bromide as the detecting fluoro- chrome (195). Blood cells were lysed, and the remaining bac- teria were stained with ethidium bromide; as few as 10 *E. coli* cells/ml were detected. Using ethidium bromide fluorescence and light-scattered signals, Cohen et al. (58) were able to

detect bacteria in 43 clinical specimens from several sources, such as wound exudates, bile, serous-cavity fluids, and bron- chial-lavage fluids, in less than 2 h, although they were unable to identify them.

An FCM method for the direct detection of anaerobic bac- teria in human feces was described by van der Waaij et al. (322), using PI for discriminating the patient's cells and ex- cluding large particles by forward light scatter. At the same time, fluoresceinated antibodies against human immunoglob- ulin A (IgA) were added to detect IgA-coated bacteria. This method allows the rapid and highly sensitive assessment of fecal floraby specific IgA-FITC fluorescence without the need to culture the samples.

Another way in which FCM can achieve direct diagnosis is by fluorescent-oligonucleotide detection. By combining rRNA- targeted fluorescent probes and 4,,6-diamidino-2-phenylindole (DAPI) for nucleic acid staining, Wallner et al. (333) showed that it was possible to detect *Acinetobacter* spp. by FCM. To date, this approach has not been used with clinical samples, perhaps owing to its methodological complexity. Nevertheless, the specificity provided by the oligonucleotide probe to identify the putative infectious agent can be taken advantage of (315), thus promising many future applications.

The use of different-sized fluorescent microspheres coated with antibodies against microbes is a new application of flow cytometry for direct diagnosis (169). This method detects the binding of specific microbes to antibody-coated microspheres by measuring the decrease in the fluorescenceemission of the microspheres due to the shading effect of microbes on both the exciting and emitting light. With different-sized fluorescent microspheres, several pathogens can be detected simulta- neously in the same sample. This approach could also be used with fungi, parasites, and viruses, as well as in infections pro- duced by combinations of these. In fact, as discussed below, a similar approach has been used for the simultaneous detection of plant viruses (137).

Fungi. With regard to yeasts, the work by Groshen et al. (122), Chaffin et al. (47), and Han et al. (126) has shown that surface antigens of *Candida albicans* can be detected by flow cytometry in conjunction with available specific antibodies. As discussed below, this approach can be used for clinical samples.

The possibility of serotyping *Candida* isolated from clinical samples emerged from the work of Chaffin et al. in 1988 (47) and Brawner and Cutler in 1989 (40). However, it was not until 1996 that Mercure et al. (219) validated the FCM serotyping procedure, using serotype A-specific antisera. According to Mercure et al., the most striking feature of this method is its reliability. Ninety-four strains isolated from patients were an- alyzed by a slide immunofluorescence assay and FCM. FCM was able to detect the presence of two different strains in a culture that was assumed to be pure and serotyped four strains whose serotypes could not be determined by slide immunoflu- orescence. Again, it was acknowledged that when a cytometer is available, the procedure is probably more cost-effective than a commercially available kit for *Candida* serotype determina- tion. Since the origin of the infecting strain(s) is often ques- tioned when clinicians encounter patients with repeated epi- sodes of *Candida* infections, FCM can help to discriminate among strains, as demonstrated in this work (219).

The diagnosis of onychomycosis based on clinical presenta- tion, culture, and microscopy is hampered by false-negative and false-positive results that confuse treatment outcomes.

Using FCM and antibodies directed against yeasts, Pierard et al. (256) identified fungal pathogens and differentiated them from nonpathogenic ones. Furthermore, the authors demon-strated that mixed infections occur, and hence the treatment for such circumstances can be established.

Parasites. The first applications of FCM to parasites in- volved a study of the cell cycle and the amounts of DNA of *Physarum polycephalum myxamoebae* (319) and the character- ization of monoclonal antibodies against membrane antigens from *Leishmania* (337). Specific clinical applications came later, when Flores et al. (99) used monoclonal antibodies, FCM, and immunofluorescence microscopy for the direct iden- tification of *Naegleria fowleri* and *Acanthamoeba* spp. in clinical specimens.

Several approaches have been developed in the last few years to detect intracellular parasites, such as *Plasmodium* (147, 153, 240–242, 250, 319, 325). Such work took advantage of the absence of DNA in erythrocytes. Thus, if the parasite is inside the cell, its DNA can be stained with specific fluoro- chromes and detected by FCM. The multiparameter analysis permitted by FCM can be used to study other characteristics, such as parasite antigens expressed by the erythrocyte (which can be detected by antibodies conjugated with fluorochromes) (54, 153) or the viability state of the parasitised cell. Further- more, the technique can be used with either fresh or fixed cells (241, 242). An important study showing the benefits of flow cytometry is that of Dixon et al. (81), who compared normal light microscopy, immunofluorescence microscopy, and FCM in the detection of *Giardia lamblia* cysts. They showed that when FCM isused in combination with immunofluorescence, a larger number of samples can be analyzed in a relatively short period and, more important, that this technique affords more consistent results than either conventional or immunofluores- cence microscopy when samples containing small number of cysts are analyzed.

Viruses. FCM allows both detection and quantification of infected cells directly in clinical samples or after inoculation and culture of the virus in cell culture.

(i) Detection and quantification of viral antigens. FCM can detect viral antigens either on the surface of or within infected cells (172). It can rapidly detect and quantify virus-infected cells using antibodies that specificallyr ecognize surface or in- ternal antigens (91, 211, 213, 334); in the latter case, perme- abilization of the cells is required. A thorough review of dif- ferent permeabilization methods, including the advantages and disadvantages of each, for viral antigen and nucleic acid de- tection has been written by McSharry (211). Direct and indi- rect fluorescent-antibody methods are used. Direct detection involves the use of fluorescently labeled antibody (labeled with FITC or phycoerythrin). In the indirect fluorescent-antibody method, unlabeled antibody is bound to infected cells, which are then incubated with fluorescence-labeled anti-Ig that binds to the first viral antibody. As previously stated,

FCM is carried out on single cells, and therefore FCM analysis of virus-infected cells is best suited to blood, bronchoalveolar lavage fluid, and urine samples (172). However, it is also possible to analyze cells from tissues that have previously been treated enzymatically (211).

Based on the potential ofFCM for multiparametric analysis, there are two key advantages to its use in studying viral infec- tion: (i) its ability to analyze several parameters in single- infected cells at the same time and (ii) its ability not only to detect but also to quantify infected cells.

These parameters may be related to particular components or events of the infected cell or components (proteins or nucleic acids) of the virus. For this reason, FCM has been a powerful tool to char- acterize the mechanisms of viral pathogenesis. Furthermore, FCM allows simultaneous detection of several viruses in a sample by using (i) antibodies to different viral antigens con- jugated to different fluorochromes, or (ii) specific viral anti- bodies conjugated to latex particles of different sizes.

As stated above, the presence of different viral antigens is detected by differences in the forward-scattered light as a consequence of the different sized particle used for each antibody. For exam- ple, Iannelli et al. (137) simultaneously detected cucumber mosaic, tomato, and potato viruses by using 3-, 6-, and 10-µm- diameter latex particles, respectively. Although this method was aimed at the detection of plant viruses, its basis could be applied to the detection of animal or human viruses in any clinical sample, such as the simultaneous detection of CMV, HSV, and HBV in organs destined for transplantation as well as in transplanted patients and coinfections in HIV-infected individuals.

Flow cytometric analysis has allowed the detection and quantification of SV40 T antigen in infected cells and moni- toring the kineticsof T-antigen expression. By means of mul- tiparametric analysis, using PI for the measurement of cellular DNA and FITC-labeled antibody for the detection of viral antigens, Lehman et al. (171, 179) related high levels of SV40 T antigen to the appearance of cells with tetraploid DNA content due to a cell cycle block at G2/M. Detection of immediate-early, early, and late CMV antigens by monoclonal antibodies permits direct diagnosis and quan- tification of CMV infection. This is a frequent complication in immunosuppressed patients, including transplant recipients and AIDS patients. In 1988, by means of FCM, Elmendorf et al. (91) detected early CMV antigen 30 min after virus adsorp- tion to fibroblasts. Thus, FCM permits the detection of viral infection earlier than does conventional immunofluorescence microscopy or the detection of cytopathic effects in cell culture (91, 289, 310).

During active infection, CMV disseminates in the blood, and viremia has been described as a major risk factor for the progression to clinical disease, particularly in allogeneic bone marrow transplant recipients (31). Accordingly, quantification of the viral load in persistently infected hosts may provide a method to predict the development of CMV disease and help to differentiate symptomatic infection from asymptomatic shedding. Preventive strategies increasingly use the CMV load as a surrogate marker for disease and initiate antiviral treat- ment based on the systemic viral load (31). Sensitive tech- niques, such as the pp65 antigenemia assay or the quantitative PCR assay, allow the detection and quantification of systemic CMV. Both assays provide a good estimation of the systemic CMV burden. Owing to the high sensitivity of these assays, CMV is also detectable in patients with asymptomatic infec- tions. However, patients with disease often have a higher viral load and can therefore be discriminated (31, 314).

The pp65 antigenemia assay determines the systemic CMV load and consists of direct staining of polymorphonuclear leu- kocytes with monoclonal antibodies against the lower matrix protein pp65 (31, 112, 314). This determination has classically been made by very difficult and time-consuming microscopic observation of immunostained cells (112). Recent studies have evaluated FCM for the direct detection and quantification of CMV antigens in polymorphonuclear leukocytes from trans- plant recipients (76, 132, 141). Measurement of pp65 CMV antigenemia by FCM overcame these problems owing to its speed and automation and showed it to be a specific and reproducible method, especially when the paraformaldehyde-methanol permeabilization-fixation method and antibody 1C3 (to late antigens) were used (141). Good agreement was found between the degree of DNA load and the level of antigenemia detected by FCM in renal transplant recipients. Although the sensitivity of the method was somewhat lower than that of the slide method, it might be sufficient to predict the disease.

Honda et al. (132) also identified specific CMV-infected cell populations in peripheral blood lymphocytes from CMV-in- fected patients by FCM. Using monoclonal antibodies directed against immediate-early CMV antigen (see above) or against several cell membrane markers to phenotype infected periph- eral blood cells from bone marrow transplant recipients, these authors developed a rapid and quantitative FCM method for the detection of immediate-early CMV antigen. The detection of CMV antigens specifically in the polymorphonuclear leuko- cytes from transplanted patients with CMV pneumonia sug- gests that the FCM antigenemia assay would be useful for predicting CMV-associated disease in transplant recipients (132). In summary, FCM offers a rapid and suitable quantifi- cation of the CMV viral load. Although systematic compara- tive evaluations of the CMV viral load using this method are needed, current data are promising.

The detection of cytomegalic endothelial cells in peripheral blood of patients is another means of monitoring active CMV infection. Through enrichment of endothelial cells in the mononuclear fraction by density centrifugation, endothelial cell-specific staining, and fluorescence-activated cell sorting of these cells, a method with 10-fold greater sensitivity than cy- tocentrifugation of the mononuclear cell fraction alone has recently been developed for quantification of cytomegalic en- dothelial cells by FCM (158). Belles-Isles et al. (24) have also suggested using FCM to monitor CMV infections by monitor- ing the CD8+ CD38+ T-cell subset in kidney transplant recip- ients; this T-cell subpopulation usually increases during active viral infections. Quantification of CD8+ CD38+ T cells by dual-color FCM in 77 kidney transplant recipients during the posttransplantation period detected high levels of CD8+ CD38+ subsets in all patients with CMV disease. Belles-Isles et al. (24) therefore concluded that the percentage of CD8+ CD38+ T cells constitutes an immunologic marker that can serve as a tool for early detection of viral diseases.

Viral antigens have also been detected by FCM for the diagnosis of hepatitis and herpesvirus infections. Quantitative and dynamic analyses of hepatitis virus markers are important in the follow-up of antiviral treatments (32). HBV surface (HBsAg) and HBV core (HbcAg) antigens in peripheral blood mononuclear cells (PBMCs) from HBV patients have been detected by FCM using antibodies (52, 278). In one study, 35 patients with HBV chronic active hepatitis and 38 out of 60 patients with acute hepatitis B (63%) expressed HbsAg in PBMC. In another work, Chemin et al. demonstrated the se- lective detection of HBsAg and HBcAg on B lymphocytes and natural killer cells from chronically HBV-infected patients (52). Hepatitis C virus (HCV), woodchuck hepatitis virus, and varicella-zoster virus were also detected by FCM in PBMCs using monoclonal antibodies to HCV core antigen (34), poly- clonal antibodies to woodchuck hepatitis virus (51), and anti- bodies to the gpI glycoprotein from varicella-zoster virus (296), respectively. Thus, FCM detection of viral antigens offers a potentially useful automated assay for the clinical diagnosis of multiple blood-borne viruses.

HSV antigens have also been detected in HSV-1, HSV-2, and human herpesvirus 8 (HHV-8)-infected cells by FCM (117, 213, 300, 347). After overnight amplification of clinical samples suspected to contain HSV, FCM detected virus 1 to 3 days before cytopathic effects were detected in cell culture (213). Sensitive FCM assays have also been recently developed to quantitate rotavirus in clinical and environmental samples (2, 19).

Finally, FCM has also been extremely useful in the study of HIV infection (174, 211, 213). By studying HIV-infected cell lines by FCM in 1987, Cory et al. (61) determined the percent- age of infected cells and the relative amount of p24 antigen per cell. These and other authors used the same assay to detect and quantify HIV-infected cells in cell cultures by monitoring p24, p17, nef, gp120, gp41,and gp160/gp41 expression (61, 62, 130). This method proved to be more sensitive and accurate for quantitative studies and faster than other methods of HIV-1 detection, such as the reverse transcriptase (RT) assay or de- termination of syncytium formation.

Detection of HIV antigens on peripheral blood mononu- clear cells by FCM is a useful method for monitoring HIV replication in vivo by monitoring the number of circulating CD4+ cells positive for p24 (63, 131, 235), p24 and nef (213), or p18 and p24 (107). In all these works, the percentage of cells expressing these antigens was statistically correlated with the clinical status of the patient. Furthermore, the authors re- ported an inverse correlation of HIV antigen-positive mono- nuclear cells and the number of CD4+ cells. Therefore, these assays are useful for rapidly monitoring disease progression in HIV-seropositive individuals and for monitoring the effect of antiviral therapy. Some studies found a lack of correlation between cell-associated antigen detection by FCM and the detection of HIV antigens in sera from HIV-seropositive indi- viduals by the standard antigen capture enzyme-linkedimmu- nosorbent assay (ELISA). As explained by McSharry et al. (213), the lack of correlation is due to a masked antigenemia in the presence of immunocomplexes, which could underestimate the amount of antigen present in peripheral blood detected by ELISA. However, in spite of being a good assay for evaluating disease progression, FCM detection of HIV antigens is not sensitive enough to detect the low levels of HIV-infected pe- ripheral blood cells in asymtomatic HIV-1-seropositive indi- viduals (213). This lack of sensitivity can be overcome, as discussed below, by coupling in situ PCR and FCM for the detection of small numbers of peripheral blood cells infected with low levels of HIV in asymptomatic individuals.

(ii) Detection and quantification of viral nucleic acids. The emergence of PCR and rPCR RT-PCR techniques has allowed the highly sensitive detection of specific viral nucleic acids (DNA or RNA) in virus-infected cells. These methods are indeed the most sensitive for the detection and characteriza- tion of viral genomes, especially in the case of rare target viral sequences (123). However, the association between the viral nucleic acid and an individual cell is lost, and therefore no information about productively infected cell populations is ob- tained by this method. FCM analysis of fluorescent in situ hybridization in cell suspension overcomes this problem (33, 173), since this assay can be coupled with simultaneous cell phenotyping (by using specific antibodies to different cell markers).

FCM detection of in situ hybridization has been used to analyze rare virus-producing cells in peripheral blood samples. HIV-1 RNA in infected cell lines was detected by fixation of the cells in suspension, hybridization with HIV-1 genomic probes labeled with digoxigenin–11-dUTP, detection with flu- orescent anti-digoxigenin antibody, and FCM analysis of the fluorescence signals thus generated (33). Link et al. (184) de- tected CMV antigen pp65 with immunoenzymatic labeling by day 4, whereas CMV-DNA was detected by PCR coupled to FCM detection of in situ hybridization 4 h postinfection on T-lymphoblastoid cells (MOLT-4). This method also detected and quantified mononuclear peripheral blood leukocytes in a patient with active CMV infection (184). Of CMV-DNA-pos- itive mononuclear peripheral blood leukocytes from patients with active CMV infection, 15% were detected by this method,
while only 0.9% were found when CMV antigens were ana- lyzed by immunoenzymatic labeling (184). Identification of specific CMV-DNA or RNA by this method, with the possi- bility of phenotyping cells by FCM, should permit latency stud- ies in CMV infections through the identification of specific cells actively replicating the virus and cells that harbor the virus in a latent state, acting as a reservoir for infection. Further- more, FCM permits these cells to be sorted for the character- ization of latency and reactivation mechanisms.

Two fluorescence in situ hybridization-FCM assays have also been developed for monitoring EBV-infected cells in blood (66). Crouch et al. were able to quantify EBV-infected cells in suspension for both the latent and replicative phases of the virus, using in situ hybridization with two different fluores- cently labeled probes (specific for each phase of EBV replica- tion) and FCM (66). This in situ hybridization-FCM assay detected one positive cell out of 9,000, which is sufficient for a diagnosis of EBV-infected cells in transplant recipients with lymphoproliferative disease.

As an alternative to conventional PCR radioactive methods, nonradioisotope FCM detection of viral PCR products was developed (342–344). Following virus-specific PCR amplifica- tion incorporating digoxigenin-labeled dUTP, labeled ampli- cons are hybridized with biotinylated probes, the hybrid DNA is captured using streptavidin-coated beads, and FCM analysis of the binding of FITC-labeled anti-digoxigenin antibodies is performed (342–344). This PCR immunoreactive bead (PCR- IRB) assay has been used for the detection and quantification of HIV-1 (342, 343) and HBV (344) viral genomes. As few as two or three copies of HIV-1 proviral DNA sequences were rapidly detected in PBMC from HIV-1-infected blood donors, a sensitivity comparable to that of the conventional radioactive detection of PCR products (342, 343). The PCR-IRB assay is a very simple, specific, sensitive, and automatic assay for the detection of specific HIV-1 amplicons. Yang et al. (343), test- ing a panel of 20 pedigreed PBMC specimens, demonstrated a perfect correlation with the results from conventional radioac- tive assays. By this method, Dorenbaum et al. (84) detected about three copies of proviral HIV DNA using primers for the long terminal repeat sequences. In a double-blind study of blood samples from 14 mother-infant pairs using the PCR-IRB assay, these authors obtained similar results to those found with the commercial Amplicor HIV-1 PCR kit. On testing 20 specimens of blood donors, with or without markers of HBV infection, PCR-IRB detected HBV DNA in a 1,000-fold- higher dilution than the infectious dose needed to produce infection in chimpanzees (344). The PCR-IRB assay proved to be specific and more sensitive than the PCR analyses involving hybridization with radioactive probes for the detection of HBV in blood. Importantly, the FCM assay avoids the use of radio- isotopes.

FCM and RT-PCR have detected gene expression in indi- vidual cells (111). Muratori et al. (225) developed an in situ RT-PCR technique using fluorescein-labeled HCV specific primers detected by FCM for the quantification and pheno- typing of HCV-infected cells in clinical blood samples. Al- though HCV infects PBMC, the small proportion of circulating infected cells is not easily detectable by conventional RT-PCR. These authors detected HCV in PBMC cells of 50% of patients with chronic hepatitis C tested; the proportion of HCV-in- fected cells ranged from 0.2 to 8.1%. Recently, a very sensitive and powerful PCR-driven in situ hybridization assay has been developed (115). This method combines the sensitivity of PCR with the specificity of in situ hybridization, allowing rapid and reproducible detection of single-copy proviral DNA or low-abundance viral mRNA in subsets of cells in suspension.

This assay employs PCR- or RT-PCR-driven in situ amplification of viral sequences in fixed cells in suspension with sequence-specific primers and digoxi- genin-linked dUTP. The product DNA is hybridized with a fluorescein-labeled oligonucleotide probe, and the cell suspen- sion is then analyzed by FCM (245). This method has been used for the detection of HIV-1 DNA and mRNA sequences in individual cells in both cell lines and cells from HIV-1-infected patients (243–245). The sensitivity and specificity of this tech- nique revealed a linear relationship for the detection of a single copy of intracellular proviral DNA over a wide range of HIV-1-infected cell concentrations (245). Re et al. (274) ana- lyzed the presence of HIV-1 proviral DNA in PBMC from HIV-infected patients at different stages of the disease by a PCR-in situ hybridization FCM assay and correlated the data with p24 antigenemia and virus isolation. p24 antigenemia correlated with the number of CD4-positive cells but was de- tected in only a very low percentage of patients with a cell count greater than 200 CD4+ T cells per liter. As stated above, detection of HIV antigens is not sensitive enough to detect the low levels of HIV-infected peripheral blood cells in asym- tomatic HIV-seropositive individuals. The virus was isolated in most patients with a T-cell count below 500 per liter but only in 4 of 14 patients with a cell count higher than 500 CD4+ T cells per liter. In contrast, the PCR-in situ hybridization FCM assay revealed detectable levels of proviral DNA in all the HIV-1-positive subjects studied, even those with a cell count higher than 500 CD4+ T cells per liter. These data underscore the potential of this assay for detecting small numbers of PBMC infected with low levels of HIV in asymptomatic indi- viduals.

In HIV-1-infected patients, plasma viral RNA levels (viral load) correlate with disease progression (105, 216, 327). Ac- cordingly, evaluation of this marker by RT-PCR is extensively used to monitor the kinetics of HIV infection and the effects of antiretroviral treatments (12). However, the RT-PCR assay does not characterize the cell populations contributing to the plasma viral load. The PCR-driven in situ hybridization method coupled to the FCM assay described above allows simultaneous phenotyping of infected cells and hence quanti- fication of HIV-1 proviral DNA or RNA molecules in specific cell populations. Patterson et al. (244) identified and quanti- fied cell subsets in the peripheral blood of HIV-1 patients expressing HIV-1 RNA by using PCR-driven in situ hybridiza- tion coupled to FCM. They found a good correlation between the FCM-determined percentage of HIV RNA-positive cells and the expected percentage of HIV RNA-positive cells on the basis of plasma viral load, with sensitivities of less than 30 copies of RNA per cell and a detection limit of 3.5% HIV-1 RNA-positive cells within a heterogeneous population.

. Simul- taneous immunophenotyping by FCM showed that a signifi- cantly higher fraction of patients with a high plasma viral load (more than 20,000 copies/ml) harbored HIV RNA-positive monocytes than did those with a low plasma viral load. Fur- thermore, the PCR-driven in situ hybridization and FCM assay permits the determination of the presence in HIV-1-infected patients of both latent and transcriptionally active viral infec- tion by detection of proviral DNA or viral mRNA. Patterson et al. (245), testing nine HIV-1-infected patients, observed a sig- nificant proportion of PBMCs infected with HIV-1,with most of the cells having viruses in a latent state (the percentage of PBMCs with proviral DNA varied from 4 to 15% whereas the percentage of PBMCs with *tat* mRNA varied from 1 to 8%). The above FCM nucleic acid detection techniques have sim- ilar sensitivity to conventional PCR, but with the added bene- fits derived from expression analysis in individual cells (inconventional PCR, nucleic acid expression is not analyzed in- dependently in each cell). Multiparametric analysis of infected cells allows the detection of single-copy proviral DNA or low-abundance viral mRNA (225, 244, 245) in specific subsets of cells that can be phenotyped at the same time. Moreover,FCM is a very useful tool to study the mechanisms of viral latency by association of different stages of the virus cycle and disease progression with the location of the virus in specific cell pop- ulations.

This can be achieved by using probes to specific mRNAs related to different viral replication stages (66). Knowledge of cell populations in which the virus is either replicating or in a latent state has important implications for our understanding of virus replication in vivo and progression to disease and hence for therapeutic treatments. Furthermore, it should be possible to sort these cells for further analysis. Double staining of viral nucleic acids together with viral pro- teins or surface markers is also possible (33, 66). In comparison with the detection of the PCR-driven in situ hybridization by fluorescence microscopy (92), in which a large number of mi- croscopic fields must be studied, FCM allows the analysis of thousands of cells in a few seconds. The speed and automation of these assays make them optimal for the rapid diagnosis of viral infections. Since these assays can also determine therel- ative number of cells bearing viral genomes and the viral load, they could be used to evaluate and monitor antiviral treat- ments. Amplification of nucleic acid sequences of viruses from cerebrospinal fluid, blood, or tissues, which are difficult to isolate by conventional diagnostic techniques, together with the detection of such nucleic acids by FCM open new possi- bilities in the diagnosis of viral infections and the character- ization of viral pathogenesis.

Serological Diagnosis

Bacteria. The identification of pathogens by microsphere immunoassays using FCM offers the specificity provided by antibodies coupled with the speed and multiparametric analy- sis provided by FCM. Although these assays can be used to directly detect microbes, they are more useful for detecting antibodies against microbes in sera obtained from patients. Generally, either a bacterial antigen preparation or the whole organism is attached to polystyrene microspheres with a uni- form diameter. The antigen-coated microspheres are incu- bated with the sera, the putative human antibodies recognize the antigen, and, in a second step, a fluorescence-conjugated antibody against human Igs is used for detection. FCM allows this assay to be completed in a short time with excellent sen- sitivity and reliability. Using this approach, Best et al. (27) detected the presence of antibodies against *Helicobacter pylori* in sera from 55 patients. These authors demonstrated that this method was as sensitive and reliable as ELISA but faster and cheaper. The simplicity of the technique and the stability of the coated microspheres make the FCM immunofluorescence as- say highly practical for serodiagnosis.

The characteristics of FCM allow the detection of more than one antigen at the same time. Thus, simultaneous detection of multiple antibodies using different-sized particles coated with different antigens or microbes would make it possible to detect multi-infection diseases. Furthermore, using several fluoro-chrome-conjugated antibodies against human Igs and differ-ently sized microspheres, FCM can gather information from each different-sized microsphere and particular fluorescence signals. Using *Brucella abortus*-coated microspheres and *Staph-ylococcus aureus* fixed cells, dual antibody detection in sera and milk from cows has been reported by Iannelli et al. (138). In this assay, antibodies against the two bacteria are identified on the basis of the altered size of *B. abortus* cells due to the microspheres.

Another use of the microsphere fluoroimmunoassay by FCM is to detect bacterial toxins. In cases where the suspicion of *Clostridium difficile* infection is high, it is necessary to con- firm the presence of toxin A in patient samples. Renner (275) reported a microsphere fluoroimmunoassay for *C. difficile* toxin A using microspheres of two different sizes. The largest one was coated with polyclonal antibody against toxin A, and the smaller one, which was fluorescent, was coated with mono- clonal antibody against toxin A. In the first step, large micro- spheres were added to the stool samples, and after incubation, smaller fluorescent microspheres were added. FCM measure- ment allowed the separation and washing steps to be omitted by gating the light scattered by the larger microspheres and measuring only the associated fluorescence from the smaller particles. Renner compared this method with the cytotoxin assay and culture of the organism from patients with *C. diffi- cile*-associated gastrointestinal disease. The results showed that the fluoroimmunoassay was less sensitive than the cytotoxin assay and culture but had the same specificity,with the advan- tage of being rapid, and, as the author stated "in laboratories with a flow cytometer, this offers an alternative method for the laboratory diagnosis of *C. difficile*-associated gastrointestinal disease." Tapp and Stotzky (313), using a similar approach to detect and track the fate of toxins from *Bacillus thuringiensis*, concluded that FCM is more sensitive and rapid than dot blot ELISA and that it is possible to process many samples easily.

The detection of antibodies with borreliacidal activity in sera from patients with Lyme disease can help in both early and late serodiagnosis. Using FCM, it is easy to detect the loss of viability of *Borrelia burgdorferi* incubated with sera from pa- tients with Lyme disease, using fluorochromes that detect the damage caused by antibodies. Callister et al. (43) used acridine orange to demonstrate this effect.

The above work demonstrates the potential of FCM as a routine technique in clinical microbiology laboratories for de- tecting the presence of antibodies against microbes inpatient sera and for reliably checking the presence of toxins in clinical samples.

Fungi. The use ofFCM and the antibodies present inpatient sera to detect fungal pathogens was first described by Libertin et al. (183) in 1984. *Pneumocystis carinii* cysts in lung homog- enates from biopsy specimens were detected by these authors using sera from patients and experimentally infected rats. Bergbrant (26), using FCM to monitor antibodies against *Pity- rosporum ovale* in sera from patients with seborrheic dermati- tis, demonstrated that there was no relationship between this microorganism and the illness.

Although the tools to directly detect antibodies against fungi inpatient sera do exist, no work validating the FCM procedure has yet been published.

Parasites.

The presence in patient sera of antibodies to any particular parasite can permit an FCM-based diagnosis of par- asitism. Martins-Filho et al. (199), using FCM on serum from patients chronically infected with *Trypanosoma cruzi*, devel- oped a sensitive method for the immunodetection of anti- trypomastigote membrane-bound antibodies. They were also able to monitor the treatment in order to establish its effec- tiveness. A similar assay was developed by Cozon et al. (64) with *Toxoplasma gondii*, using fixed tachyzoites and specific conjugates for different human Ig heavy chains. They were able to quantify the amounts of IgM, IgG, and IgA antibodies in patient sera by measuring the amount of fluorescence bound to tachyzoites. The authors stated that the method might offer a major improvement in cost-effectiveness per sample (especially when a large number of tests is used)
compared with routine immunofluorescence assays by fluorescence microscopy and further stressed that it could be fully automated.

Viruses.

Some methods have been routinely used to detect specific antibodies to viral antigens. Among these techniques are ELISA, complement fixation, indirect immunofluores- cence microscopy, and Western blotting. In addition, the de- tection and quantification of antibodies to viral antigens can be carried out by FCM. This technique has been used to detect and quantify antibodies to CMV (209), HSV-1 and HSV-2 (46, 209), HCV (187, 210, 279), and HIV-1 (100, 118, 133, 290, 294).

Most of these viral antibody quantifications use a micro- sphere-based immunoassay and FCM. In this assay, polysty- rene microspheres attached to viral antigens are used as a support for viral antibody detection by FCM. For the simulta- neous detection of two or more viruses, different-sized micro- spheres, each coated with a specific viral antigen, are used. The assay has the advantage of simultaneous detection of multiple antibodies with high analytic sensitivity. Simultaneous detec- tion and quantification of antibodies to CMV and HSV was achieved by McHugh et al. (209) using this method. Using particles of different sizes coated with p31, gp120, p24, and gp41 antigens from HIV-1, Scillian et al. (290) were able to detect and quantify the specific antibodies. An FCM immuno-fluorescence assay (FIFA) with high sensitivity and specificity was developed by Sligh et al. (294) to detect antibodies to HIV-1 by using HIV-1-infected cell lines. . The cells are incu- bated with the sera to be tested, and incubation with an FITC- conjugated anti-human Ig and FCM allows the quantification of HIV-1 antibodies in the sera.

Based on this assay, Folghera et al. (100) developed a FIFA for the quantitative determina- tion of HIV p24 in HIV-1-infected cells and used the reduction in HIV-1 p24 antigen expression in these cells to determine the neutralizing-antibody titers in human sera (100). This method also allowed the rapid detection and monitoring of antibodies to native and recombinant human HIV-1 envelope glycopro- teins following gp160 immunization (118). A new serological assay, the recombinant FIFA, was later described (133) for the early detection of HIV-1 antibodies. In this assay, antibodies in sera are evaluated by FCM for binding to the HIV-1 recom- binant insoluble forms of proteins Gag-p45, Gag-gp41, and gp160 expressed in insect cells by a baculovirus expression system. The sensitivity of this method permits earlier detection of antibodies after initial infection than for enzyme immuno- assays, with a reduction in the “window” period,i.e., the time between initial infection and the time of seroconversion, a parameter which is critical in infection from blood transfusions (133).

The humoral immune response to HCV has been evaluated in patients with chronic hepatitis (187). Antibodies to HCV core and NS3 antigens have been quantified using immunoas- say beads and FCM (210) in blood donors. The microsphere assay resulted in increased sensitivity (fivefold higher than that of reference methods) of HCV detection and resolved a sig- nificant proportion of indeterminate samples. A fast FCM as- say that measures the neutralization of the binding of recom- binant HCV E2 envelope protein by antibodies to human cells has also been described (279). This method permits study of the natural immunity to HCV and should be useful in the development and validation of vaccination protocols. To conclude, the investigations of Best et al. (27) and Ian- nelli et al. (138), among others, offer the possibility of perform- ing FCM serodiagnosis in an elegant, rapid, cheap, and precise manner. The technique is simple and can be used for many pathogens (including viruses), with an additional possible ad- vantage of detecting more than one microorganism in a single sample. The use of FCM in clinical microbiology laboratories would allow detection times and costs to be reduced. At present, however, it is not in general use and the setting up of such protocols can be fairly time-consuming.

ANTIMICROBIAL EFFECTS AND SUSCEPTIBILITY TESTING BY FLOW CYTOMETRY

The first experiments in which FCM was used to study the effects of antimicrobial agents in prokaryotes were carried out at the beginning of the 1980s (136, 302, 304, 306). In the 1990s, there were interesting advances in this field from microbiology laboratories, and the number of scientific articles addressing the antimicrobial responses of bacteria (including mycobacte- ria), fungi, and parasites to antimicrobial agents increased con- siderably. The development of FCM in combination with fluo- rochromes permits the assessment of individual viability and functional capacity (membrane potential and metabolic path- ways) within microbial populations. This allows investigators to explore the possibility of performing susceptibility testing within the applications of FCM. Also, the introduction of this technique in clinical laboratories for routine susceptibility test- ing has been proposed, since this approach can be performed reliably in just a few hours. Several examples of the study of the antimicrobial effect and susceptibility testing by FCM are shown in Table 2.

Available data clearly demonstrate the utility of this tech- nique to also assay viral susceptibility. Since the pioneer works of Rosenthal et al. (280) and Pauwels et al. (247) on the use of FCM to study the effect of antiviral agents on herpesvirus and HIV, FCM has been used by several groups, particularly those studying these viruses and CMV. The detection and quantifi- cation of viral antigens and viral nucleic acids in combination with antibodies or nucleic acid probes, respectively, or any other cellular parameter associated with viral infection have been used in FCM protocols for the in vitro evaluation of antiviral-drug activities. In addition, the possibility of on-line monitoring of the antiviral treatments ex vivo has also been explored. The latter approach has also been applied to bacte- rial and fungal infections; rapid and sensitive protocols for the evaluation of microbial responses to antimicrobial agents are also available.

In the following sections, the possible uses of FMC in anti- microbial susceptibility testing are discussed according to the type of pathogen involved.

Antibacterial Agents

Standardized methods for performing in vitro susceptibility testing of bacteria in clinical microbiology laboratories are widespread (98). Qualitative and/or quantitative results are given on the basis of the size of the zone of inhibition or MIC. Despite the automation of MIC-based broth microdilution sys- tems, an 18- to 24-h incubation period is usually needed before antimicrobial activity can be quantified. Recently, fluorogenic, turbidometric, and colorimetric technology has reduced sus- ceptibility testing to 4 to 6 h (83, 98). However, unless the bacterial inoculum is quantified, only the bacteriostatic effect, i.e., the MIC, is generally tested by clinical laboratories. The MBC, which reflects the bactericidal effect of antimicrobial drugs, is rarely determined. This requires calculation of bacte- rial counts, generally expressed as CFU, which involves bacte- rial culture dilutions and subcultures. In addition, neither MIC nor MBC determinations consider the heterogeneity of bacte- rial populations. Similarly, postantibiotic and subinhibitory concentration effects (106), which offer pharmacodynamic data based on the antimicrobial activity, are rarely determined in clinical laboratories since these determinations (190) are te- dious and time-consuming.

FCM has proved to be very useful for studying the physio- logical effects of antimicrobial agents on bacterial cells due to their effect on certain metabolic parameters (membrane po- tential, cell size, and amount of DNA). In addition, FCM is a reliable approach for susceptibility testing, offering results in terms of the bactericidal or bacteriostatic effect (86, 108, 198, 262, 331).

The studies by Martinez et al. (198) and Steen et al. (302) in 1982 on the antimicrobial effects on bacteria assayed by FCM are now considered classic. They demonstrated that the effect of β-lactam antibiotics on *E. coli* can be detected by measure- ments of light scattering and DNA content after 10 min of incubation with the drug. Using FCM, Steen et al. (302) showed the effect of several antibiotics that inhibit protein synthesis (chloramphenicol, erythromycin, doxycycline, and streptomycin) on *E. coli* with the fluorescent DNA probe mith- ramycin (305). Since then, FCM has been used to measure the effects of different antimicrobial agents on bacteria (Table 2).

Realizing the potential of FCM in this field, several compa- nies have developed new products to rapidly perform antibiotic susceptibility testing in clinical microbiology laboratories. We have used one of these products (*Bac*-light; Molecular Probes) to analyze the reliability of this test in clinical microbiology. We worked with two strains of *Enterococcus*, one sensitive to vancomycin (*E. faecalis* ATCC 29212) and other resistant (*E. faecium* U2A1), with a *vanA* element responsible for its van- comycin resistance phenotype. In addition, as a control we included an *E. coli* isolate showing intrinsic resistance to van- comycin. The above-mentioned products are kits with two fluo- rochromes that permit the detection of live and dead cells. Bacterial cells were incubated with and without vancomycin for 2 h at the respective MICs of this drug (Fig. 5). Antimicrobial effects were detected by measuring the variations in fluores- cence due to dead cells within 3 h of incubation with the antibiotic. Another advantage of FCM (Fig. 5), as mentioned above, is the visualization of the heterogeneity of the response of the cells to the antimicrobial agent. This heterogeneity is detected by determining the presence of subpopulations that are less susceptible to the antimicrobial agent treatment. Da- vies and Hixson have recently reported similar results (69). Therefore, it is feasible to use FCM and fluorescenceprobes to perform rapid testing of the susceptibility of bacteria to a panel of antimicrobial agents before choosing the treatment. More- over, resistant subpopulations can be effectively detected, rep- resenting an interesting advantage ofFCM that can be suitably exploited in clinical studies (331).

Measurement of bacterial susceptibility.

By using fluores- cence probes, FCM measures the light scattering of cells in suspension and fluorescence of cellular contents or metabolic activity. Thus, the activity of antimicrobial agents can also be described in terms of changes in cell shape and in fluorescence (262, 329, 330). In this sense, Walberg et al. (329, 330), and others (108, 200) showed that light scattering is a useful pa- rameter of antimicrobial effects, irrespective of their mecha- nisms of action. This was demonstrated with two different drugs: ceftazidime, which acts on the bacterial envelope, and ciprofloxacin, whose target is a gyrase. Nevertheless, the same authors (329, 330) used both fluorescence detection and light- scattering analysis when susceptibility testing was performed directly in clinical samples in order to distinguish bacterial cells from debris and other components.

Two major categories of fluorochromes have been used to study antimicrobial effects and short-term antimicrobial activ- ity :

: (i) DNA- and RNA-labeling dyes and (ii) metabolic or protein-binding probes, which include membrane potential- sensitivity dyes (262) (Table 2). The choice of these substances in a particular protocol is related to their excitation and emis- sion properties and to the mechanism of action of the antimi- crobial. Mithramycin with ethidium bromide (DNA-staining dyes) were the first probes used for susceptibility testing by FCM (302) and are still in use (331). These probes estimate viability in an indirect way. After staining, the antibacterial activity in antimicrobial-exposed cells is measured as a result of the ploidy in organisms undergoing filamentation (unable to generate independent cells after division). This has been per- formed with β-lactam antibiotics, which affect cell membrane integrity but induce an increase in nucleic acid contents in growing but not dividing cells. DNA ploidy has also been studied by Durodie et al. (86) in *E. coli* with sub-MIC concen- trations of β-lactam antibiotics.

PI, a fluorochrome that intercalates into double-stranded nucleic acid, is a more appropriate stain than ethidium bro- mide for susceptibility testing by FCM (262). Because PI is a nonpermeable dye that is excluded by viable cells, measure- ment of the loss of viability produced by antimicrobial agents or other compounds is possible. For example, Gant et al. (108) studied the effect of several classes of antimicrobial agents, such as an aminoglycoside (gentamicin), β-lactams (ampicillin, mecillinam, and cefotaxime), and a DNA gyrase inhibitor (cip- rofloxacin), on the integrity of the outer membrane of *E. coli* as measured by the uptake of PI. This method allows differenti- ation among antibiotics with different cell wall enzyme targets due to its effects on cell light-scattering characteristics and its effect on the integrity of the outer cell membrane of *E. coli*, measured simultaneously.

Other new nucleic acid-staining fluorochromes that provide more intense fluorescence and resolution have also been used to test antimicrobial activity (189, 203). SYTO-13 and SYTO-17 label both DNA and RNA (127). These stains were used by Comas and Vives-Rego (59) in combination with oxo- nol to assess the effects of gramicidin, formaldehyde, and sur- factants on *E. coli* viability and membrane potential. Another fluorochrome is acridine orange (202), which stains DNA and RNA at the same time but with different wavelength emissions. Therefore, variations in the relative amount of each nucleic acid in the presence of antimicrobials can be evaluated. Also, acridine orange penetration depends on plasma membrane integrity, and so the method can also be used to measure alterations in plasma membrane permeability produced by the action of antimicrobial agents.

Bacterial cells respond to different antimicrobial agents by decreasing or increasing their membrane potential. Moreover, cell death and/or membrane damage brings about a collapse of the electrical potential. Membrane potential can be effectively measured by FCM using potential-sensitive probes (262). These dyes are normally positively charged and accumulate inside the cell, rendering it fluorescent. As the cell depolarizes, the dye is redistributed or the electronic environment changes and the fluorescence produced by each fluorochrome molecule decreases. Some of these probes belong to the carbocyamide group of dyes, which includes 3,3,-dipentyloxocarbocyanine iodine [DiOC5]. This dye was previously used to measure membrane potential in mammalian cells by FCM (291). Or- do ez and Wehman (237) applied DiOC 5 to assess the suscep- tibility of *S. aureus* to penicillin G and oxacillin. The results were comparable to those obtained by conventional suscepti- bility tests but were obtained 90 min after addition of the antimicrobial, as opposed to the usual overnight period.

Rhodamine 123 and oxonol [bis(1,3-dibutylbarbituric acid) trimethine oxonol] are other membrane potential probes used to assess drug effects by FCM (59, 150, 310). Their accumula- tion inside bacterial cells depends on the difference in charge between the two sides of the plasma membrane. Rhodamine 123 is a cationic lipophilic dye. Because it is not readily taken up by gram-negative organisms (157, 205), it is used only for gram-positive organisms. Rhodamine 123 is accumulated in the cytosol by cells with an active transmembrane electrochem- ical potential. In contrast, oxonol is an anionic lipophilic dye that can enter the cell only when the membrane has become depolarized. Therefore, fluorescence-emitting cells have a low membrane potential; the higher the decrease in membrane potential, the higher the intensity of oxonol fluorescence (67, 78, 157, 189, 203, 237).

Since the same fluorochrome can be used in different stud- ies, versatility is an essential property. Oxonol has been used for susceptibility testing of different classes of antimicrobial agents in both gram-positive and gram-negative organisms (Table 2). Mason et al. (200) used this dye to analyze mem- brane damage caused by gentamicin, a protein synthesis inhib- itor, and ciprofloxacin, a DNA gyrase inhibitor. Suller et al. (310) used it to study the effect of penicillin, methicillin, and vancomycin, all of which interfere with bacterial envelope syn- thesis. In addition,Jepras et al. (150) used oxonol to study the action of azithromycin, cefuroxime, and ciprofloxacin on *E. coli*. Another advantage of oxonol is that it can be added directly to the liquid culture, avoiding any pretreatment steps that may cause perturbations in bacteria and interfere with the effect of the antimicrobials. Furthermore, oxonol is nontoxic to bacterial cells, unlike cationic dyes (79).

Measurement of antimycobacterial drug susceptibility.

Flu- orescein diacetate (FDA) (a nonfluorescent diacetyl fluores- cein ester that becomes fluorescent upon hydrolysis by cyto- plasmic esterases) staining and FCM have been used by Norden et al. (231) for the FCM susceptibility testing of *My- cobacterium tuberculosis*. In contrast to the delay in obtaining data in current antimycobacterial drug susceptibility testing, results were available within 24 h. In addition, using FDA, Bownds et al. (36) demonstrated that mycobacterial multipli- cation was not required to measure antimycobacterial drug activity, as is the case with traditional methods. *Mycobacterium avium*, *Mycobacterium gordonae*, *Mycobacterium fortuitum*, and *Mycobacterium marinum* treated with different concentrations of ciprofloxacin, kanamycin, and other tuberculostatic agents were easily differentiated from untreated cells after 6 and 24 h of incubation (36). Using the same protocol, Kirk et al. (164) determined the susceptibilities of 35 clinical isolates of *M. tuberculosis* to several concentrations of isoniazid, rifampin, and ethambutol in 24 h. To avoid manipulator infection risks, Moore et al. (222) have developed a variation of the above method using paraformaldehide to inactivate *Mycobacterium*. In this case, the time needed to obtain susceptibility results increased to 72 h.

A different approach has been explored to use FCM to detect mycobacterial growth inhibition under antimicrobial pressure based on mycobacterial microencapsulation in aga- rose gel microdrops. FCM was used to monitor colony growth by evaluating the intensity of auramine staining after different times of incubation in the presence or absence of rifampin and isoniazid. With this system, growth inhibition was demon- strated after 1 to 3 days, and, indeed, the presence of an isoniazid-resistant subpopulation was observed (283).

Intracellular bacterial pathogens.

Antimicrobial suscepti- bility testing by FCM has also been used for intracellular pathogens such as *Rickettsia tsutsugamushi* (160). Conventional susceptibility methods for this organism include direct micro- scopic counting of Giemsa-stained cells and mouse protection assays. Both methods are slow, labor-intensive, and expensive in comparison with FCM screening. Kelly et al. (160) evaluated the in vitro effectiveness of doxycycline and azithromycin against both doxycycline-sensitive and -resistant *R. tsutsuga- mushi* strains. Inhibitory concentrations were assayed by mea- suring the reduction in the number of infected cells deter- mined by fluorescence emission of stained *R. tsutsugamushi*. This method allowed screening of numerous isolates for their susceptibility to a variety of antibiotics that was faster than conventional techniques. The correlation between FCM screening and conventional susceptibility testing was high.

Recently, Dessus-Babus et al. (75) developed a specific and reproducible technique for the susceptibility testing of another intracellular pathogen, *Chlamydia trachomatis*. The results for susceptibility to doxycycline, ofloxacin, and erythromycin obtained by FCM were compared with those obtained using the standard method of determining the minimal chlamydicidal concentration by microscopie examination. The assay was performed on 13 clinical isolates after infection of McCoy cells and incubation for 25 h with and without the antimicrobial agent. An FITC-conjugated monoclonal antibody to the major outer membrane protein of *Chlamydia* was used to stain chla- mydial inclusions. Calculation of the 50% inhibitory concen- tration (IC50) by FCM allowed a more objective and precise evaluation of antimicrobial activity than did reading of micro- scopic inhibitory concentrations. The values obtained were re- producible and could be applied to compare the results ob- tained with different drugs.

Postantibiotic effect.

The presence of a transient inhibition of bacterial growth after prior exposure to antimicrobial agents (postantibiotic effect) was first described by Bigger (30). Although this effect is responsible for a persistent inhibition of bacterial cells after short-term exposure to an antimicrobial agent (190), the mechanism(s) through which this effect is exerted remains obscure (106). These data are generally used to define antimicrobial dosing, e.g., single daily doses, as in the case of the new fluoroquinolones and aminoglycosides (101, 307). Traditionally, studies of the postantibiotic effect have been based on bacterial counts (190), and only a few authors have explored the possibility of using other approaches (65). By detecting changes in the impedance of the culture media, Baquero et al. (18) were able to measure bacterial growth. This parameter is related to the metabolic activity of the cell pop- ulation and may be considered equivalent to measuring viable cells in a given population. Other authors have used optical density,carbon dioxide generation, intracellular ATP measure- ments, and the rate of [3H]thymidine incorporation to assess microbial growth (65). Recently, FCM has been used to suc- cessfully determine the postantibiotic effect (119, 309).

This technique has the advantage of studying the heterogeneity of bacterial populations and particular metabolic events during the postantibiotic-effect period. Gottfredsson et al. (119) stud- ied microbial sizes and nucleic acid contents in *E. coli* and *P. aeruginosa* populations during the exposure of these microor- ganisms to antimicrobial agents as well as during the postan- tibiotic effect period (after drug removal). Aliquots of bacterial growth were stained with PI at regular time intervals during and after drug exposure. Different effects could be observed during the post-antibiotic period. In general, the postantibiotic effect induced by β-lactam antibiotics and ciprofloxacin is char- acterized by the appearance in the population of enlarged cells with increased nucleic acid contents whereas rifampin induces a decrease in the size and nucleic acid contents of the micro- organism. Both effects would have been missed if only bacterial counts had been monitored. Suller et al. (310) also reported the heterogeneity of bacterial populations during the postan- tibiotic-effect phase in *S. aureus* cultures previously exposed to methicillin. Despite the observation of very few variations in the numbers of CFU during this phase, a continuous decline in membrane potential and respiratory activity was found using FCM. Again, these effects would have been missed ifFCM had not been available.

Antifungal Agents

Interest in antifungal agents and resistance to these antimi- crobials has redoubled during the last decade.

Invasive fungal infections have emerged as major causes of morbidity and mortality due to the increasing numbers of immunocompro- mised and HIV patients. Nevertheless, antifungal susceptibility testing in clinical laboratories is currently performed to a far lesser extent than is susceptibility testing of antibacterial agents. Unlike bacteria, standardized methods and interpret- able guidelines have only recently been proposed for antifun- gal agents, and even then they remain controversial (253). Different definitions of breakpoints for susceptibility testing may occur as a result of the absence of consensus-standardized MIC techniques for different antifungal drugs (165), hinder- ing the interpretation of results and protocol standardization. The broth dilution methods for yeast susceptibility testing of the National Committee for Clinical Laboratory Standards (NCCLS) are labor-intensive and require 48 to 72 h of incu- bation. Alternative methods including the E-test (253), the Bioscreen microdilution method (323), and a colorimetric sys- tem (94) have also been proposed.

The FCM assay has been proposed as a reliable alternative method to study antifungal activity. Moreover, FCM has also been used for yeast susceptibility testing (120, 163, 234, 252, 259–262, 270, 336). The results obtained with FCM agreed with those obtained by NCCLS methods and were available in only 24 to 32 h or even in 9 h (163, 233). Moreover, the definition of end points for antifungal treatment by FCM is probably easier (252).

There are several studies based on FCM. In 1990, Pore et al. (259) developed an FCM assay to study the susceptibility of *C. albicans* to ketoconazole, amphotericin B, and 5-fluorocy- tosine. Using both PI and rose bengal, the time required for susceptibility determinations decreased from 24 to 48 h to only 9 h. Sensitivity and reliability were greater than in the standard NCCLS broth dilution method. The assay could also be ap- plied to other pathogenic yeasts such as *Candida tropicalis*, *Candida parapsilosis*, *Candida lusitaniae*, *Candida krusei*, *Can- dida guilliermondii*, *Torulopsis glabrata*, and *Rhodotorula rubra* (262) and *Crytococcus neoformans* (261). O'Gorman and Hopfer (233) used ethidium bromide and FCM to study drug susceptibility in yeasts, measuring the effects of amphotericin B on *Candida* species after 8 h of incubation. Carter et al. (45) performed similar studies using different fluorochromes to de- tect dead and living cells (PI and FDA) and changes in mem- brane potential (oxonol) and intracellular ion concentrations (Indo-1 and Fluo-3).

Unfortunately, no relationship between the antifungal agents and the ion concentration could be es- tablished, since the ion-sensitive probes were unable to stain yeast cells. The authors reported alterations in oxonol fluores- cence after 20 min of incubation with amphotericin B, although with other fluorochromes, such as PI, the response time was longer (8 h). Differences in the detection times of the various fluorochromes depend on the parameter measured by the flu- orochrome. Since amphotericin B mainly affects the plasma membrane potential, oxonol will first detect this effect. By increasing the amphotericin B incubation time, the plasma membrane will be affected to a greater extent, which will per- mit the entrance of PI.

In 1994, using FDA, Green et al. (120) showed that FCM was a better and faster method than conventional ones for determining and comparing the antifungal activities of com- pounds with different modes of action. Since then, several studies have shown that FCM can be effectively used for anti- fungal susceptibility testing. Ord n ˜ez and Wehman (237) mea- sured the variations in the membrane potential of different *Candida* species with DIOC5 when cells were incubated with amphotericin B. Sensitivity or resistance was determined after 30 min in the presence of the antifungal agent. Moreover, two recent articles have reported the effectiveness of PI in testing antifungal susceptibility by FCM (163, 270). **o**

The results of both studies were comparable to those obtained with the reference NCCLS macrodilution method during a period ranging from 2 to 24 h. The antifungals studied were amphotericin B, flucon- azole, and itraconazole. Recently, Peyron et al. (252) have compared FCM with the NCCLS broth dilution method for amphotericin B. Oxonol was used to measure the variations in membrane potential. From the dose-response curve obtained for each isolate, the concentrations at which the fluorescence intensity was reduced by 50, 80, and 90%, i.e., the IC50, IC80, and IC90, respectively, were calculated. Regression analysis revealed that the best agreement with the M27-T NCCLS document was obtained with the IC80 end point. Thus, FCM can be effectively used for routine susceptibility testing since this endpoint is currently used in clinical laboratories to dis- tinguish susceptible from resistant isolates. Moreover, the re- sults were highly reproducible and were obtained within 1 h.

Also, susceptibility studies of *Pneumocystis carinii* have been performed using FCM and PI (223). Although significant ad- vances have been made in susceptibility testing for filamentous fungi (73), to our knowledge FCM has not yet been used.

Factors Affecting Bacterial and Fungal Susceptibility Testing

Several authors have underscored the importance of differ- ent factors that can affect bacterial susceptibility testing by FCM. Although such factors do not imply a clear limitation in the FCM assay, they may give misleading results. Knowledge of these factors is critical for the implementation of methods to avoid false resistance and/or false susceptibility results. These factors have been studied with bacteria, but similar consider- ations could also be applied to other organisms, such as fungi or parasites.

The organism itself can affect susceptibility testing by FCM. Because different bacterial species and occasionally even dif- ferent strains of the same species show different abilities to take up membrane potential-sensitive dyes, this has been sug- gested to affect susceptibility testing by FCM (201). The ability of rhodamine 123 to stain cells differs between gram-negative and gram-positive organisms (205), possibly due to the highly charged nature of gram-negative bacterial lipopolysaccharide. Moreover, enhanced efflux mechanisms resulting in extrusion of the internalized dye may decrease the amount of dye present inside the cells of certain bacteria and thus give mis- leading results (95, 151, 220).

Dye concentrations and staining times can also affect sus- ceptibility testing by FCM (59, 108, 201). For example, the labeling properties of oxonol in *E. coli* are related to dye concentrations (201). The effects of valinomycin were better defined at oxonol concentrations of 10 than 0.5 mg/liter. Nev- ertheless, a high dye concentration can produce cytotoxic ef- fects (259). Therefore, staining protocols for susceptibility test- ing should be carefully established and appropriate controls should be used.

Another important issue is the cell preparation. For in- stance, permeabilization is used in certain FCM protocols. This procedure allows certain nonpermeable dyes to cross the cy- toplasmic membrane, while permeable dyes cross more readily. Therefore, incubation times must be changed. Pretreating gram-negative bacteria with 1 mM EDTA improves the uptake of the membrane potential dye rhodamine 123, allowing the detection of antibiotic effects on the membrane potential of gram-negative bacteria. However, such pretreatment can affect the physiological parameters of bacterial viability and indeed the membrane potential (79, 205).

Afixation step is mandatory in DNA staining of bacteria with nonpermeant stains. This can be achieved by 70% ethanol treatment (37), but two steps of centrifugation are required, thus increasing the processing time and loss of cells. An alternative to ethanol fixation is the cold shock procedure, which has been used with *E. coli* cells (151). Permeabilization of exponentially growing *E. coli* in- volves placing the cells in ice-cold EDTA (10 mm) in phos- phate-buffered saline (pH 7.4) containing 4 g of azide per liter. The cold shock procedure was recently used by Walberg et al. (331) in combination with ethidium bromide and mithramycin. This seemed to be as efficient as ethanol fixation for detecting the effects of β-lactams and quinolones but not for detecting the effect of gentamicin.

The use of surfactants has also been proposed to render cells permeable to fluorescence dyes but can affect the FCM results (59). Because these agents may exert biocidal activity, they could interfere with the antibacterial effect of the antimicrobial agent being tested. Also, these agents may generate indepen- dent particles (debris) which are detectable by the cytometer as light scatter signals (59). Sometimes these signals are propor- tional to the surfactant concentration and can be attributed to micelle formation and cellular aggregation. Nevertheless, by using cationic surfactants with null biocidal activity, this pitfall has been circumvented (59).

The antimicrobial drug concentration should be carefully controlled since different concentrations may give different results. In early studies, Steen and coworkers used concentra- tions exceeding the MIC values for bacteria (302, 303) but other authors used lower and even sub-MIC concentrations (87, 198). Over the last few years, antimicrobial effects have been studied at clinically achievable concentrations (58, 200, 237, 329, 330). Gant et al. (108) tested antimicrobials by FCM at concentrations equivalent to conventional MICs. In this study, the exposure time of bacteria to the antimicrobial agents prior to the FCM assay was also critical for the demonstration of the inhibitory action.

The inoculum and bacterial growth phase are other factors that may affect FCM results. Comas and Vives-Rego (59) used stationary-phase instead of exponential-phase cells mainly for two reasons: (i) stationary-phase inocula are used in conven- tional antimicrobial assays, and (ii) stationary-phase cells re- main stable longer than do exponential- or dead-phase cells. These authors observed a progressive decline in rhodamine 123 uptake during the growth phase, whereas exponential or stationary populations did not take up oxonol, which was in-corporated only in the dead-phase. In general, bacterial and yeast inocula are slightly higher in FCM susceptibility assays than in conventional assays. With this approach, resistant sub- populations can be better detected.

Like any other technique, FCM requires work and time in the setting up of protocols. The factors affecting the results are the same as those affecting conventional susceptibility proto- cols plus those intrinsic to FCM, such as aggregation, cell size, and autofluorescence. Once these are delineated, FCM can be routinely used in antimicrobial susceptibility testing. The pos- sibility of using simple commercial kits, such as the one de- scribed in Fig. 5, will encourage the use of FCM in clinical microbiology laboratories.

Antiparasite Susceptibility

Drug resistance among parasites is an increasingly recog- nized phenomenon, particularly among different *Plasmodium* species (88, 242). Clinical failure, rather than laboratory re- sults, is taken as indicative of a drug resistance process. Unlike bacteria and yeasts, testing of parasites for susceptibility to antimicrobial drugs is not generally performed in clinical lab- oratories due to technical difficulties and the absence of stan- dardized methods.

Interestingly, different authors have used FCM to study the effect of antiparasitic drugs and explore the possibility of routine susceptibility testing. For example, Azas et al. (15) used FCM to assess the effects of pentamidine, allopurinol, and amphotericin B on *Leishmania infantum* me- tabolism with PI to measure viability and DIOC5 to detect membrane potential alterations. Moreover, using FCM and rhodamine 123, Maarouf et al. (192) demonstrated that paro- momycin rapidly affects the mitochondrial activity of *Leishma- nia donovani*, lowering rhodamine 123 fluorescence. They sug- gested that the decrease in rhodamine 123 fluorescence could be used to detect sensitivity to drugs in this parasite. An FCM multiparameter approach was developed by Basselin et al. (21) to study the differences between pentamidine-resistant and -nonresistant *Leishmania*. They detected variations in mito- chondrial membrane potential and lipid metabolism in penta- midine-resistant cells.

Pattanapanyasat et al. (242), working with a drug-resistant *Plasmodium* strain, described an antiparasite drugsusceptibil- ity method using FCM. Using several iron chelators as anti- parasite drugs, FCM measured their effects on *Plasmodium* viability by detecting parasites inside the red blood cells. They demonstrated the importance of iron for parasite growth and showed that FCM provides a simple and reliable means of antimalarial drug susceptibility testing. Analyzing environmental samples, Bamdad et al. (16) used FCM to test the uptake and efflux of several polycyclic aromatic hydrocarbons in *Tetrahymena* spp. in an attempt to obtain evidence of a resistance mechanism.

As stated above, FCM has already been used to study the effects of antiparasite drugs. Despite this, its application in routine practise does not seem to be as common as in the study of antibacterial and antifungal agents, perhaps for socioeco- nomic reasons rather than because of scientific criteria.

Anti viral Susceptibility

Although antiviral in vitro susceptibility testing is not routine in the clinical microbiology laboratory, the development of rapid and accurate tests is essential for the screening of new antiviral compounds and antiviral susceptibility studies. From a clinical point of view, the emergence of antiviral resistance increases the necessity for rapid, automated, and objective assays. Different methods commonly used to test viral susceptibility include plaque reduction assays, virus yield reduction assays, assays based on inhibition of the virus-induced cyto- pathic effect, and assays for the detection of viral nucleic acids and viral antigens. These conventional methods are labor-in- tensive and time-consuming.

Because viral antigens, nucleic acids, or any viral parameter related to viral infection can be quantified by FCM, the antiviral susceptibilities of clinical iso- lates or laboratory strains can be rapidly determined. FCM also permits rapid screening and assessment of new potential antiviral compounds and their mechanisms of action. In a recent review, McSharry (212) has described the applications of FCM to (i) the screening of antiviral compounds active against human CMV, HSV, and HIV and (ii) the performance of drug susceptibility testing for clinical isolates. Since both FCM studies of antiviral susceptibility in clinical isolates or laboratory strains and studies of antiviral activities or antiviral mechanisms of action are based on the same principles and techniques, we will review both in this section by virus type.

HSV. Rosenthal et al. (280) developed a rapid and easy FCM method to screen the anti-HSV activities of several an-tiviral agents. The method was based on HSV-induced changes in cellular DNA content measured by the binding of PI to the DNA. The lower the antiviral activity, the larger was the DNA amount and the more intense was the PI fluorescence (280).

The activity of antiviral drugs against HSV-1 (300), HHV-6 (276) and varicella-zoster virus (295, 296) has also been as- sayed by FCM by measuring the inhibition of specific viral antigen expression for each drug with antibodies against virus antigens. For example, Pavic et al. (248) developed a quanti- tative FCM method measuring gpC expression to determine the susceptibility of HSV-1 to acyclovir, ganciclovir, and fos- carnet; this was done by measuring the amount of antibody against gpC binded to infected cells. The results were com- pared with those obtained by the standard cytopathic effect inhibitory assay. A very interesting conclusion was reached: while both methods gave identical qualitative results, the range of IC50 detected for acyclovir-resistant strains by FCM was much broader than that detected by the cytopathic effect in- hibitory assay for the same isolate.

Therefore, it is possible to detect resistant viral subpopulations within phenotypically characterized sensitive populations by using FCM. In view of the emergence of drug-resistant viral subpopulations after long-term treatment with acyclovir, this finding is of great interest in the clinical setting, since it should be possible to detect not only resistance but also the emergence of resistance in a patient.

CMV. FCM has been used for rapid in vitro evaluation of anti-CMV agents (185, 227, 295). Neyts et al. (227) used FDA to measure the cytoplasmic esterase activity after CMV infec- tion. Differences in FDA fluorescence allowed these authors to differentiate between CMV-infected and -uninfected cells and to evaluate several antiviral agents. Snoeck et al. (295) moni- tored the anti-CMV activity of several phosphonyl methoxyal- kylpurines and pyrimidines by FCM and DNA hybridization. Recently developed by McSharry et al. (214, 215) and Kesson et al. (162), an FCM assay differentiated gancyclovir-sensitive and -resistant strains of human CMV clinical isolates and lab- oratory strains by quantification of human CMV immediate- early and late antigens with specific monoclonal antibodies. In all these studies, the accuracy of the FCM assay was confirmed by parallel testing by the plaque reduction assay (PRA). There was an excellent correlation between IC50 determined by FCM and the values determined by PRA, although the FCM assay gave a broader range of IC50 than the PRA did (214, 215). Furthermore, the effect of ganciclovir was detectable by FCM at 96 h postinfection instead of the 7 days required for PRA.

HIV. In 1987 Pauwells et al. (247) used an FCM assay to quantitatively measure the inhibition of viral antigen expres- sion by 3,-azido-2,,3,-dideoxythymidine (AZT) and 2,,3,-dideoxycytidine (ddCyd) in HIV-1-infected human T4 lympho- cyte cells. The anti HIV-1 activity of a broad range of compounds has subsequently been studied using FCM (113, 166, 193, 208, 218, 224, 236, 284–288, 341). Using FCM and MT-4 cells, Schols et al. (287) monitored the cell death pro- duced by HIV-1 infection using FDA and also observed the effect of AZT. A dose-dependent viral inhibition was observed by FCM that was comparable to the results obtained by the more tedious and time-consuming conventional methods. FCM determination was able to monitor the effects of antiret- roviral treatment regardless of the status of p24 antigen ex- pression in infected cells (62, 208).

FCM has also evaluated the effect of inhibitors of HIV-1 proteases and other antiretroviral drugs on the expression of cell-associated viral proteins in treated peripheral blood lymphocytes and cell lines (166, 218, 341). AZT and 2,,3,-dideoxyinosine (ddI) inhibit the establish- ment of HIV-1 infection in CD8+ cells derived from peripheral blood lymphocytes as shown by FCM analysis using anti-gly- coprotein 120 and anti-CD8 monoclonal antibodies to quantify the percentage of CD8+ infected cells (CD8+ gp120+) (218).

Using a coculture system of chronically HIV-1 infected monocytes and noninfected CD4+ T lymphocytes, it is possible to use FCM to study viral dissemination and hence the inhibi- tion of dissemination in the presence of antiviral compounds (224). Recently, Macchi et al. (193), using a similar coculture system of PBMC isolated from normal adults and the chroni- cally HTLV-1-infected MT-2 cell line, measured the levels of viral DNA and RNA by PCR and viral core p19 expression by FCM. The inhibitory effect of AZT on virus transmission to PBMC cells was demonstrated (193). Coculturing uninfected JM cells and HIV-1 chronically infected H9 cells and using two-fluorescence FCM analyses, Bridges et al. (41) demon- strated the dose-dependent effect of the drug 6-*O*-butanoyl castanospermine on virus production as measured by the de- creases in cell conjugate formation between normal and chron- ically infected cells.

A different way to study the effect of antiviral agents is to quantify the expression of viral genes using gene reporters. A green fluorescent protein reporter system in which the gene encoding this protein is under the control of the HIV-1 long terminal repeat promoter was described for the rapid and simple quantification of HIV infection (85). In the presence of HIV-1, expression of the reporter gene is strongly induced, and FCM measurement of the relative fluorescence in cells allows reliable quantification of infected cells. Recently, Gervaix et al. (113) obtained a stable T-cell line expressing a similar green fluorescent protein construct. This stable cell line offers a very rapid, easy, and accurate method for monitoring HIV-1 infec- tion and evaluating new antiretroviral compounds. Rapid de- termination of drug susceptibility to HIV-1 RT and protease inhibitors as well as other antiretroviral drugs was also per- formed by these authors.

Poliovirus.

The infection of human fibroblasts by poliovirus leads to morphological changes related to cell size and granu- larity in infected cells, which can be detected by FCM. The analysis of this induced cytopathic effect, as stated above, has been extensively used to monitor viral infection, and FCM allows perfect monitoring of these changes. Furthermore, [Ca2+]i can be monitored simultaneously. As shown in Fig. 6, [Ca2+]i increases from 2 to 3 h postinfection and 90% of the cells increase their [Ca2+]i by the 5 h postinfection. These effects on cell size and the increase in [Ca2+]i in infected cells must be a consequence of viral gene expression, since such modifications are not observed when cells are incubated with cycloheximide, guanidine, or Ro 09-0179 compounds (145). This is a good example of multiparametric analysis of viral infection and also highlights the potential of this technique for studying antiviral activity.

Antiviral drugs: mechanisms of action.

Understanding the mechanisms of action of antiviral drug is important for the design of new antiviral compounds and the improvement of existing ones. Some examples of FCM used in studies of anti- viral mechanisms of action with HIV are explained below. Since FCM has permitted the study of virion-cell binding through specific receptors, it allows the characterization of antiviral drugs that inhibit this process, as demonstrated by Schols et al. (284). They used human anti-HIV serum in an FCM assay to detect the HIV-1 virion binding to the cell membrane, analyzing several compounds for their ability to inhibit it. While AZT did not affect viral binding, heparin, dextran sulfate, and pentosan polysulfate completely inhibited the process. Suramin, an inhibitor of RT, partially blocked HIV-1 binding. Subsequently, these authors showed that these compounds which inhibited HIV-1 binding to MT-4 cells block the interaction between the gp120 and the CD4 receptor (286). Using an FCM assay based on a DNA-staining protocol which reveals differences in DNA contents in HUT-78 cells persis- tently infected with HIV-1 and CD4+ MOLT-4 cells, these compounds were shown to suppress syncytium formation and destruction of CD4+ cells.

Of note, Oravecz et al. (236) later studied the role of cell surface proteoglycans in chemokine- mediated anti-HIV-1 activity in T cells and macrophages. FCM revealed that digestion of cell surface proteoglycans, including heparin sulfate, prevented the binding of RANTES to PM1 (CD21H) cells (a human T-cell line selected for high CD26 expression). Finally, using FCM, Schols et al. (288) measured the effect of AMD3100 (a prototype of bicyclams that is a new selective inhibitor of HIV-1 and HIV-2 replication) on the binding of a specific CXCR4 monoclonal antibody to SUP-T1 cells. The drug inhibited this binding, indicating an interaction with the CXC chemokine receptor CXCR4 (coreceptor for T-tropic viruses). However, AMD3100 did not inhibit the bind- ing of CC chemokine macrophage inflammatory protein, a ligand of the chemokine CCR5 receptor (coreceptor for M- tropic viruses).

Summarizing, a close correlation is always found between the FCM assay and conventional susceptibility methods. Fur- thermore, FCM decreases the time required for antimicrobial susceptibility studies and also allows multiparametric analysis, meaning that more information is obtained from the same sample. Finally, it can be used to gain further insight into the mechanisms of action of antimicrobial agents.

MONITORING OF INFECTIONS AND ANTIMICROBIAL THERAPY

The evaluation of patient responses to antimicrobial treat- ments is usually based on clinical findings. Nevertheless, dif- ferent microbiological studies are frequently performed to as- sess the efficacy of antimicrobial treatments (152). In vitro susceptibility tests provide information about the concentra- tion at which a specific microorganism is susceptible. However, in vitro results may not correlate with the response to in vivo therapy since other factors affect this susceptibility (152). Among these factors are the immunological status of the pa- tient, the viral or bacterial load, and the possibility of selecting antibiotic-resistant strains as a consequence of the antimicro- bial treatment. The last point is particularly interesting in viral infections. Virus-infected patients can shed multiple viral sub- populations with potentially different susceptibilities, which hinders the determination of the correct treatment. For these reasons, monitoring of antimicrobial treatments ex vivo will in the future be a powerful tool to evaluate the response to treatment or to show whether the treatment should be modi- fied. This is especially interesting for infections in which resis-tant strains might emerge during the course of treatment, particularly in HIV infection, for which simultaneous multith- erapies have been developed. From the therapeutic point of view, it would be desirable to detect, as early as possible, evolving resistant strains in a patient in order to decide on the most suitable treatment.

As mentioned above, Cohen and Sahar (58) described a FCM method to detect bacteria in body fluids and exudates (wound exudates, bile, serous-cavity fluids, and bronchial-la- vage fluids). These authors also demonstrated that the antimi- crobial effects of amikacin could be detected directly in bacte- ria present in body fluids. A total of 43 clinical samples were prepared for FCM by centrifugation, filtration, resuspension, and the addition of growth medium including 100 mg of ami-kacin per liter. Light scattering and nucleic acid staining with ethidium bromide were used to assess the bacterial response. The results obtained in less than 2 h were in agreement with those obtained by conventional methods (plate counts and turbidometry). Susceptibility to amikacin was detected in 1 h in 92% of 13 bacterium-positive specimens. Furthermore, with respect to yeasts, Martin et al. (197) developed a PI-based FCM bioassay that measures amphotericin B concentrations in human serum, detecting its fungicidal effect on *C. albicans* cells in only 5 h.

More recently, Walberg et al. (331) studied antibiotic sensi- tivity testing in polymicrobial infections. They designed an in vitro model for the FCM detection of heterogeneous drug responses in exponentially growing *E. coli* and *Klebsiella pneu- moniae* cells. Bacteria were incubated with ampicillin, fixed, and stained with a nucleic acid-specific fluorochrome. Discrim- ination of heterogeneous populations was demonstrated, as well as the differentiation between susceptible and resistant subpopulations. The detection of resistant subpopulations is crucial in the monitoring of antimicrobial treatments, since these populations may become predominant under antibiotic pressure. Preliminary results have also been obtained with other members of the *Enterobacteriaceae* and *Pseudomonas aeruginosa* (331). Although this in vitro model has not yet been used for the ex vivo assessment of infections, this approach should make it possible to monitor polymicrobial infections and theirrespective treatments.

The viral load is commonly used as a marker of disease progression (105), and antiretroviral therapy has therefore been proposed in asymptomatic patients with a plasma viral RNA load of more than 30,000 copies/ml (44). Inhibition of viral replication induced by anti-HIV drugs is generally asso- ciated with an increase in the number of CD4+ cells and a reduction in plasma HIV-1 RNA levels and disease progres- sion. FCM has been used to monitor HIV-1 antiretroviral therapy, mostly by measuring the viral load by PCR and eval- uating CD4 counts by FCM in plasma PBMC and lymph node mononuclear cells (170, 266, 269, 312). Other constituents of the peripheral blood of AIDS patients have been used to monitor the effect of antiviral agents ex vivo by FCM. For example, the number of dendritic cells in the peripheral blood of AIDS patients decreases progressively and, quantification by FCM of the increase in dendritic-cell num- bers inpatients given AZT therapy can be used to monitor the response to antiviral treatments (246).

Fluorescence-labeled human leukocyte antigen tetrameric complexes are obtained in vitro by refolding of the heavy chain, and hence β2-microglobu- lin and specific peptides can be used to quantify HIV-1-specific cytotoxic T lymphocytes in HIV-infected patients by FCM (233). An inverse correlation between HIV-specific cytotoxic T-lymphocyte proportion and plasma viral load, as well as a decrease in the number of HIV-1-specific cytotoxic T lympho- cytes, was found after 6 months of treatment of patients with a triple combination of antiretroviral therapy.

peripheral blood by PCR- driven in situ hybridization and FCM (244, 245), phenotyping of virus-infected cells, and simultaneous quantitation of CD4+ cells offer a very practical method of using FCM to monitor antiretroviral treatments in HIV-1-infected patients. The rela- tionship between disease progression and the predominant infection of specific cell subpopulations remains to be fully elucidated, but FCM opens new possibilities for the rapid monitoring of these parameters in antiretrovirus-treated pa- tients.

CONCLUDING REMARKS AND FUTURE PERSPECTIVES

Cohen and Sahar (58) pointed out in 1989 that despite the rapid and automatic analysis provided by FCM, the introduc- tion of this technique in clinical microbiology laboratories was limited by the cost of the instrumentation and the need for well-trained operators. Since then, FCM devices have become user-friendly, the number of applications has been expanded, instrument software has been improved, and costs are gradu- ally decreasing. Furthermore, another key characteristic of FCM is its versatility: the same machine can be used to per- form direct diagnosis, antimicrobial susceptibility testing, se- rum antibody detection, and even microbial identification.

Today's flow cytometers allow the analysis of several cell functions in a rapid and multiparametric fashion (several thou- sand cells in few seconds and a minimum of five parameters per cell). At the same time, it is possible to perform qualitative and quantitative analyses of samples. Thus, FCM is a suitable technique for the study of heterogeneous populations (68, 186) under circumstances of infections due to the presence of mul- tiple microorganisms (292) or for the study of the effects of treating microorganisms with antimicrobial agents (119, 309, 330), since each particle is analyzed individually. As stated above, its main advantages are simplicity, reliability, speed, and the possibility of detecting simultaneous infections.

Monitoring different parameters in clinical samples during the course of an illness allows an assessment of the effect of therapy in patients. This information is very important since the effectiveness of antimicrobial therapy can be measured on-line by FCM. By monitoring the patient's progress via FCM, it should also be possible to detect emerging resistant strains, hence permitting suitable modifications in treatment.

The use of flow cytometers in clinical microbiology should therefore be more of a current issue than a future perspective. For microbial diagnosis, FCM has been used primarily for the detection of viruses in blood and bacteria in urine samples (a flow cytometer specific for urine has been developed) (97, 175), saving time and labor; however, when high sensitivity is needed, visual microscopic review should be performed (167). FCM should be extended to other human samples, particularly sterile body fluids and those with low microbial counts.

To our knowledge, heterogeneity has not yet been studied in the clinical setting. In cases of mixed infections or infections produced by different bacterial subpopulations, such as the persistence of different *P. aeruginosa* morphotypes in bronchial secretions from cystic fibrosis patients (74) or the presence of *H. pylori* subpopulations with different pathogenic determi- nants in the gastric mucosa (321), FCM can help to provide the correct treatment.

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FCM may also be useful for discriminating microbial variants of recognized clinical impact, i.e., small- colony re- variants, which show different morphologies and sponses to antimicrobial agents from the parent strains (R. Cantn, personal communication).

To optimize the cost-benefit ratio of FCM in clinical micro- biology laboratories, automation is necessary. This implies adapting flow cytometers to robotics for automatic sample analysis and developing software to automate analysis of the results. This kind of software is currently used in hematological studies, and hence its adaptation to susceptibility testing, for example, should not be too difficult. The robotics are more complex, but if clinical laboratories create the need, companies should show interest in developing the necessary instruments.

The increasing availability of commercial kits to test antimi- crobial susceptibility by FCM in bacteria and fungi (127) points to the future importance of this application.

. Moreover, the ability of FCM to measure individual cells, in contrast to traditional susceptibility testing methods that analyze the entire population, would lend impetus to the spread of this technique. FCM antimicrobial susceptibility protocols will probably be- come crucial for the detection of resistant subpopulations, a potential clinical concern. This additional information provided by FCM could be useful to clinicians for the selection of the appropriate antimicrobial therapy in very short times, re- ducing the time during which the patient is without treatment. In addition, this approach can be beneficial in preventing the emergence and dissemination of resistance. From this review, many technical approaches based on FCM currently in use in basic research can be "exported" to clinical microbiology. Nev- ertheless, prior to the routine application of FCM to suscep- tibility testing, the standardization of FCM protocols is man- datory. This would involve the establishment of susceptibility criteria with specific fluorochromes based on NCCLS criteria. The work of Ramani et al. (270), Peyron et al. (252), and Kirk et al. (164), among others, in which FCM susceptibility criteria are compared with NCCLS criteria demonstrates that the use of FCM in clinical microbiology is more than a mere possibil- ity. Today's ubiquitous presence of flow cytometers should help clinical microbiologists to progressively incorporate FCM into their standard protocols.

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BODY FLUIDS EXAMINATION

FIRST EDITION
COURSE NO. 178

EDITED BY
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PREFACE

THIS IS THE FIREST EDITION OF TUMOR MARKERS. IT IS ATEACHING BOOK WHICH IS DIRECTED AS APOST GRADUATE COURSE. IT IS ORGANIZED BY CLINICAL PATHOLOGY DEPARTMENT FOR POST GRADUATED STUDENT S IN CLINICAL PATHOLOGY OR OTHER ESPACIALISTS.

I ACKNOWLEDGE AND THANK ALL STAFFS IN CLINICAL PATHOLOGY DEPARTMENT FOR THEIR EFFORT AND I HOPE VERY MUCH THAT, IN THIS EDITION, I HAVE MANAGED TO MAINTAIN THE STANDERED OF CONTENT, PRESENTATION AND ACCESSIBILITY.

IN-DEPTH COVERAGE OF THE MORE ESTABLISHED SUBJECTS OF TUMOR MARKERS BOOK INCLUDING : THE CELLULAR AND LIQUID CONSTITUENTS CHARACTERISTICS OF BLOOD , CHARACTERISTICS FEATURES OF NORMAL AND ABNORMAL BLOOD CELL FORMATION AND MATURATION ,

THE STAGES OF LEUCKOCYTE DEVELOPMENT, THE ORDER OF MATURATION AND SERIES AND CHARACTERISTICS OF EACH STAGE WITH VARIATION , OCCURRENCE OF LEUCKEMIA AND ITS CLASSIFICATIONS AND TYPES , THE APPROPIATE SIGNS AND SYMPTOMS FOR THE STAGE OR TYPE OF LEUCKEMIA .

ALSO MOST COMMON TUMOR MARKERS AND ELISA TEST USED FOR ITS DETECTION IN BLOOD ARE WELL COVERED. THESE TOPICS HAVE BECOME IMPORTANT IN TEACHING TUMOR AND ITS BIOMARKERS AS APART OF CLINICAL PATHOLOGY. I BELIEVE I HAVE ACHIEVED HIGH LEVEL OF ACCESSIBILITY AND EASY READING.

KHALID MOHAMED

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7- COPPER

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WATER METABOLISM

1-WATER CONSTITUTES ABOUT 45-70 % OF TOTAL BODY WEIGHT .

2- BODY FLUIDS ARE DIVIDED IN TWO COMPARTMENTS:

(A)- INTRA CELLULAR (ICF), WHICH WHICH CONSTITUTES 65-75 % OF TOTAL BODY WATER AND 35-45 % TOTAL BODY WEIGHT .

(B)- EXTERA CELLULAR FLUIDS (ECF) CONSTITUTES ABOUT 25 % OF TOTAL BODY WATER AND 15 % OF TOTAL BODY WEIGHT .

3-)- EXTERA CELLULAR FLUIDS (ECF) FOUND IN 3 LOCATIONS :

(A)- INTRA VASCULAR (PLASMA)

(B)-INTRASTITIAL (LYMPH)

(C) – TRANS CELLULAR (IT INCLUDES : CSF ,JOINT FLUIDS ,PERITONEAL PERICARDIAL, PLEURAL AND LIQUID IN INTESTINE).

4-DECREASED TOTAL BODY WATER LEADS TO DEHYDERATION

5-DEHYDERATION IS MANIFESTED BY :

(A)-DECRASED BODY WIEGHT

(B)LOSS OF SKIN ELASTICITY

(C)-DRYNESS OF MUCOUS MEMBRANE

(D)-RETRACTIONOF EYES OF THE EYES

6- ELECTROLYTES AND WATER MOVES FREELY BETWEEN THE ICF AND ECF .

MOVEMENT OF WATER IS DEPENDANT UPON ON HYDROSTATIC PRESSURE AND OSMOTIC PRESUURE OF ECF PROTIENS

7- OSMOTIC PRESSURE OF ANY FLUIDS IS DIRECTLY RELATED TO THE NUMBER OF PARTICLES IN A UNIT OF SOLVENT AND IS MEASURED BY OSMOLE OR MILLIOSMOLE .

8- OSMOLE IS THE MOLECULAR WEIGHT OF THE SUBSTANCE MULTIPLIED BY THE NUMBER OF IONS INTO WHICH IT DISSOCIATES .

9- THE NORMAL OSMOLALITY OF ECF IS ABOUT 300 MOSM /KG

10-ECF CONTAINS SODIUM NA , CHLORIDE CL AND BICARBONATE HCO3

11- ICF CONTAINS K POTTASIUM ,MG MAGNESIUM ,ORGANIC PHOSPHATES AND PROTIEN.

12-SERUM OSMOLALITY CAN BE ESTIMATED BY USING THE CONCETRATION OF NA SODIUM , AND K POTTASIUM AS FOLLOW :

MOSM / KG = 2(NA +K) +BUN/2.8+GLUCOSE/18

13-SERUM OSMOLALITY IS USED AS AMEASURE OF ECF OSMOLALITY .

14-HYPER OSMOLALITY IS FOUND IN CASE OF :
(A)-HYPERNATREMIA
(B)-ACCUMLATION OF ENDOGENOUS SOLUTES SUCH AS GLUCOSE ,KETONE BODIES , AND UREA
15- HYPOOSMOLALITY IS FOUND IN CASE OF HYPONATERMIA
16-OSMORECEPTORS SENSE CHANGES IN OSMOLALITY . WITH HYPER OSMOLALITY (HYPONATREMIA) , OSMORECEPTORS STIMULATES VASOPRESSIN OR ADH SECRETION FROM THE PITUITARY GLAND AND STIMULATE THIRST . THIRST IS STIMULATE BY AS ALITTLE AS

1-2 % CHANGES IN OSMOLALITY .THE END RESULTS IS WATER RETENTION BY THE KIDNEY AND INCREASED WATER INTAKE . INCREASES IN FREE WATER WILL THUS WILL REDUCE SODIUM CONCENTERATION ,OPPOSITECHANGES OCCUR WITH HYPOOSMOLALITY.

17-BARORECEPTORS ARE SENSITIVE TO CHANGES IN EFFECTIVE CIRCULATING VOLUME (ECV) THE ECV IS THAT PART OF THE EXTRACELLULAR FLUID THAT IS IN THE ARTERIAL SYSTEM AND IS EFFECTIVELY PERFUSING THE TISSUES . IT USUALLY VARIES DIRECTLY WITH ECF VOLUME .

WITH HYPOVOLEMIA (DECREASED ECV), BAROECEPTORS STIMULATES THE RENIN-ANGIOTENSIN SYSTEM , THE END RESULT BEING MINERALOCORTICOID (ALDOSTERONE) RELEASE FROM THE ADRENAL CORTEX.

ALDOSTERONE INCREASED ABSORPTION OF NACL AND PROMOTES THE EXCRETION OF POTASSIUM AND HYDROGEN IN THE DISTAL TUBULES OF THE NEPHRON , NACL RETENTION PROMOTES WATER REABSORPTION , THIS CORRECTING THE HYPOVOLEMIA . HYPOVOLEMIA ALSO STIMULATES THIRST (A DECREASE IN ECV OF 7-10 % IS REQUIRED FOR STIMULATION). OPPOSITE CHANGES OCCUR WITH HYPER VOLEMIA

ANTIDIURETIC HORMONE

ADH ACTS ON CHIEF CELLS IN COLLECTING TUBULES , INCREASING PERMEABILITY OF THE TUBULES TO WATER AND UREA . ADH ACTS VIA ADENYLE CYCLASE WHICH OPEN CHANNELS IN THE TUBULES ,CALLED AQUA-PORES , FACILITATING WATER ABSORPTION .ADH IS SECRETED BY HYPOTHALAMUS IN RESPONSE TO OSMOTIC (MAINLY DETERMINED BY NA SODIUM) AND NON OSMOTIC (VOLUME DEPLETION ,NAUSIA ,FEAR,ANXIETY,PAIN,EXERCISE AND DRUGS) STIMULI.

SEVERAL THINGS INHIBIT THE ACTION OF ADH ON RENAL TUBULES AND THUS AFFECT CONCENTRATING ABILITY .

THESE INHIBITORS OF ADH INCLUDE :

(1)-HYPOKALEMIA

(2)-HYPERCALCEMIA

(3)- CORTICOSTEROIDS

(4-)-PROSTAGLANDIN E

IN ANY OF THE ABOVE CONDITIONS URINE CONCENTRATING ABILITY IS IMPAIRED FOR REASONS OTHER THAN PHYSIACL LOSS OF FUNCTIONAL NEPHRONS .

(1- SODIUM)

SYNONUM : SERUM SODIUM NA +

SPECIMEN : SERUM OR PLASMA (HEPARINE) I ML .

REFERENCE VALUE : 136-145 MEQ/LITRE ; SI UNITS : 136-145 MMOL/LITRE

METHODS : ION-SELECTIVE ELECTRODE ,SPECTROPHOTOMETER ,FLAMEPHOTOMETER .

DESCRIPTION :

1-SODIUM IS THE MAIN CATION OF EXTERACELLULAR FLUIDS WHERE AHALF OF TOTAL BODY SODIUM IS PRESENT .MOST OF THE REST SODIUM IS PRESENT IN BONES IN STABLE FORM .

2- SODIUM HAS TWO MAIN FUNCTIONS :

A-REGULATION OF ACID BASE BALANCE

B-PLAY AN IMPORTANT ROLE IN REGULATION OF OSMOTIC PRESSURE

3-THE QUANTITY OF SODIUM IS CONTROLLED BY SODIUM INTAKE AND LOSS.

4- HORMONAL CONTROL :

A-IT IS PEFORMED BY ALDOSTERONE HORMONE ,WHICH WHICH SECRETED BY ADRENAL S GLANDS : ITS ACTION IS PERFORMED ON THE RENAL TUBULES WHERE IT CONTROLS EXCRETION AND REABSORPTION OF SODIUM .

B-MOST OF SODIUM PRESENTED TO RENAL TUBULES IS REABSORBED (APPROXIMATELY 90 %) UNDER CONTROL OF ALDOSTERONE HORMONE .

C-IF THERE IS AN EXCESS OF BODY SODIUM ,ALDOSTERONE SECRETION IS REDUCED AND SODIUM IS EXCRETED THROUGH KIDNEYS :BUT IF THE SODIUM IS REDUCED ,ALDOSTERONE SECRETION AND PRODUCTION IS INCREASED AND SODIUM IS ALMOST COMPLETELY REABSORPED .

N.B SODIUM REABSORPTION REQIURES AN EQUIVALENT PASSAGE OF HYDROGEN OR PUTASSIUM IONS IN THE OPPOSITE DIRECTION .

D-EXCRETION OF SODIUM :

IT IS DONE THROUGH :

1-MAINLY KIDNEYS THROUGH URINE.

2-SWEAT .

3-SECRETIONS OF DIGESTIVE TRACTS .

4-THROUGH STOOL.

HYPONATREMIA

DECREASE IN PLASMA SODIUM CONCENTRATION IS CALLED HYPONATERMIA
IT OCCURS MOST FREQUENTLY BECOUSE OF EXCESSIVE SODIUM LOSS THROUGH THE FOLLOWING ROUTS :

1-DIGESTIVE TRACT :

A-DIARRHEA b – VOMITING

2-URINARY TRACT :

A-RENAL DISEASES RENAL FAILURE b– USING OF DIURETICS-

D- DIABETES MELLITUS AND HYPER GLYCEMIA .

3-EXCESSIVE SWEATING WITHOUT ADEQUATE SODIUM INTAKE .

4- EXCESSIVE EXUDATION FROM BURNS OR OPEN WOUNDS WHEN SODIUM INTAKE IS INDEQUATE .

5-ADRENOCORTICAL INSUFFICIENCY (ADDISON S DISEASE).

6-LIPEMIA IN WHICH LIPIDS DISPLACES ASIGNIFICANT VOLUME OF LIQUID PHASE OF PLASMA MAY RESULT IN ALOW PLASMA SODIUM ,SIMPLY BECOUSE SMALLER VOLUME OF LIQUID PHASE .

6-CONGESTIVE HEART FAILURE .

7-ADMINISTERATION OF HYPOTONIC SOLUTION SUCH AS 5% DEXTROSE .

9-METABOLIC ACIDOSIS .

10-INSUFFICIENT INTAKE .

HYPERNATREMIA

1-HYPERNATREMIA IS ARARE CONDITION .

2-IT OCCURS WHEN THERE IS LOSS OF FLUIDS CONTAIN ASMALL AMOUNT OF SODIUM AS IN DIABETES INSPIDUS .

3-EXCESSIVE SODIUM INTAKE WITH LIMITED LIQIUD INTAKE .

4-REDUCED GLOMERULAR FILTERATION RATE AS IN RENAL FAILURE.

5-HYPER ALDOSTERONISM .

2- POTASSIUM

SYNONUM : SERUM K+

SPECIMEN: SERUM OR PLASMA HEPARINE I ML .

REFERENCE VALUE : 3.9 – 5.8 % MEQ/L .

METHODS : ION –SELECTIVE ELERCTRODE, FLAMOPHOTOMETER, SPECTROPHOTOMETER .

DESCRIPTION :

1-POTASSIUM IS THE MAN CATIONS IN THE CELLS OR INTRACELLULAR

2- FUNCTION OF POTASSIUM ARE :

A-CONTROL OF MUSCULAR ACTIVITY

B- CONTROL NEURAL EXCITABILITY

3-EXCRETION OF POTASSIUM IS DONE THROUGH:

A-KIDNEY

B-STOOL

C-SWEAT

D- DIGESTIVE FLUIDS

4-ALDOSTERONE FACILITATES EXCRETION OF POTASSIUM AS IT INCREASE SODIUM REABSORPTION BY PROMOTING EXCHANGE OF SODIUM FOR POTASSIUM .

POTASSIUM IS PRESENT IN MORE HIGH CONCENTERATION INTRACELLULAR ,SO THAT NECROSIS OF TISSUES OR DESTRUCTION OF RBCS CAUSES HIGH SERUM POTASSIUM LEVEL .

HYPO KALEMIA :

1-DIARRHEA AND VOMITTING ESPECIALLY WHEN REPLACEMENT FLUIDS IS POTASSIUM (K+)FREE .
2-IATROGENIC AS IN CASE OF PROLONGED ADMINSTERATION OF MINERALOCORTICOID.
3-HYPERALDOSTERONISM
4-ALKALOSIS ,POTASSIUM DECREASES BECOUSES IT ENTERS INSIDE THE CELLS EXCHANGING WITH HYDROGEN IONS AND ALSO INCREASE EXCRETION THROUGH URINE.
5-BICARBONATE THERAPY
6-DIURETIC TREATMENT
7-RENAL TUBULAR ACIDOSIS
8-PROLONGED ANOREXIA
9-EXCESSIVE SWAETING
10-CHRONIC RENAL FAILURE
11-INSULIN TREATMENT IN KETOACIDOTIC DIABETES (WILL CARRY POTASSIUM INTO THE CELLS ALONG WITH GLUCOSE).

HYPER KALEMIA

1-ACUTE RENAL FAILURE WHERE THERE IS ANURIA OR OLIGOURIA

2-HYPOADRENOCORTICISM (ADDISONS DISEASE)

3-USING OF POTASSIUM SPARCING DIURETICS

4-INCREASE INTAKE (AS POTASSIUM CHLORIDE (KCL) USED IN CONGESTIVE HEART FAILURE)

5-ACIDOSIS

6-DIFFUSE CELL DEATH SECOUNDARY TO SHOCK ,CIRCULATORY STASIS CHEMOTHERAPY

7-FACTITIOUS (ERRORS DURING COLLECTION OF SAMPLE):

a-HEMOLYSIS –b-DELAYED SEPARATION OF CLOTTED BLOOD (DUE TO POTASSIUM LEAKAGE) –c- COLLECTION IN POTASSIUM HEPARINE.

CYSTATIN C

CYSTATIN C

SPECIMEN: SERUM OR PLASMA (HEPARIN)
METHODOLOGY: IMMUNOASSAY
REFERENCE VALUE: 0, 53 - 0, 95 mg /L

Description

Description :

1- CYSTATIN C IS ACYSTIENE PROTIENASE INHIBITOR .

2-ASMALL MOLECULE THAT IS PRODUCED BY NUCLEATED CELLS .

3-SERUM CYSTATIN C IS FILTERED OUT OF BLOOD BY THE KIDNEYS AND THAT SERVES AS AMEASURE OF KIDNEYS FUNCTION.

4- ITS MOLECULAR MASS ALLOWS IT TO BE FREELY FILTERED BY THE GLOMERULAR MEMBRANE IN THE KIDNEYS.

5-SINCE IT IS FORMED AT ACOSTANT RATE AND FREELY FILTERED BY THE HEALTHY KIDNEYS.

6-CYSTATIN C IS AGOOD MARKER OF RENAL FUNCTION.

7-SERUM CONCENTERATIONS OF CYSTATIN C ARE ALMOST TOTALLY DEPENDANT ON GLOMERULAR FILTERATION RATE (GFR) .

8-AREDUCTION IN (GFR) CAUSES ARISE IN THE CONCENTERATION OF CYSTATIN C.

9-CYSTATIN C IS ABETTER MARKER OF THE GLOMERULAR FILTERATION RATE AND HENCE OF KIDNEY FUNCTION THAN CREATININE WHICH WAS THE MOST COMMONLY USED MEASURE OF KIDNEY FUNCTION.

10- THE LEVELS OF CYSTATIN C ARE INDEPENDENT OF WEIGHT AND HEIGHT ,MUSCLE MASS,AGE, (OVER AYEAR OF AGE) ,SEX,AND FACTORS WHICH HAVE BEEN DOMENSTERATED TO AFFECT CREATININE VALUES,IN ADDITION,ARISE IN CREATININE DOES NOT BECOME EVIDENT UNTIL (THE GFR) HAS FALLEN BY APPROXIMATELY 50%.

11- MEASUTEMENTS CAN BE MADE AND INTERPRETED FROM ASINGLE RANDOM SAMPLE.

12- CYSTATIN C MAY IS USED AS AN ALTERNATIVE TO CREATININE AND CREATININE CLEARANCE TO SCREEN FOR AND MONITOR KIDNEY DYSFUNCTION IN THOSE WITH KNOWN OR SUSPECTED KIDNEY DISEASES.

13- IT MAY BE ESPECIALLY USEFUL IN THOSE CASES WHERE CREATININE MEASUREMENT IS NOT APPROPRIATE: FOR INSTANCE IN THOSE WHO HAVE LIVER CIRRHOSIS, ARE VERY OBESE, ARE MALNOURISHED OR HAVE AREDUCED MUSCLE MASS.

14- MEASURING CYSTATIN C MAY ALSO IS USEFUL IN THE EARLY DETECTION OF KIDNEY DISEASE WHEN OTHER PARAMETERS MIGHT STILL BE NORMAL, ESPECIALLY IN THE ELDERLY.

THANK YOU

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تحت تحرير وإشراف أ.د خالد محمد رئيس القسم
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محمدمحمودمحمديوسف وهيبه
ماجستير البكتيريا والمناعة والفطريات
معهدبحوث الصحة الحيوانية
خاص بمحمدوهيبه

THE TOPIC TODAY

Renal failure

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
28109091800516/2004578801966

Renal failure is a state in which the body's metabolism is disturbed as a consequence of dysfunction of the kidneys, characterized by numerous manifestations, which are attributable to the imbalance between the rate of production of various chemical substances by the body and their rates of the excretion by the kidneys.

Kidneys become unable to perform its regulator role of the electrolytes, acid-base balance and excretion of waste products.

(Syndrome of renal insufficiency)

There are two characteristics syndromes of renal failure:-

1-acute renal failure.

2-chronic renal failure.

They Are So Two Distinct , And Each One Consider Alone .

1-Acute Renal Failure :

Definition :

It Means Acute Suppression Of Kidney Filtration Resulting In Rapidly Increasing Azotemia , With Or Without Oliguria .

Acute Renal Failure Can Be Divided Into Two Phases :

1-Oilguric Phase .

2-Diuretic Phase .

1-Oilguric Phase Which Is Characterized By :

A-Sever Decrease Of Urine Volume .

B-Increased Level Of Urea And Creatinine .

C-Decreased Serum Na+ And Hyper Kalemia.

D-Sediments Shows Protein , Red Cells ,Epithelial Cells And Casts.

2-Diuretic Phase :

Begins 2 Weeks Post Onset , Gradual Increase Of Urine Volume Indicating Nephrons Recovery (Continues For 7-10 Days).

Causes Of Acute Renal Failure :-

1-Pre Renal Causes
(Vascular Diseases).
2-Renal Causes .
3-Post Renal Causes .

1-Pre Renal Causes (Vascular Diseases):-

Sudden Vasoconstriction Of Renal Arteries In The Most Important Cause Of Acute Renal Failure.

Vasoconstriction Occurs In The Following Condition:-

A-Sudden Decrease In Blood Volume Due To Hemorrhage.

B-Cardiac Failure:

C-The Presence Of The Large Quantities Of Free HB And Myoglobin.

D-Certain Completion Of Pregnancy As Eclampsia ,Concealed Accidentals Hemorrhage And Abortion.

C-Postoperative.

:++++2-Renal Causes : It Includes

A-Glomular Diseases:

*-Acute Glomerulonephritis Caused By Salmonella ,Shigella And Toxoplasma.

*-Pyelonephritis With Rapidly Necrosis Caused By Coryne Bacterium Renale.

*-Systemic Lupus Erythematosis.

*-Membranous Glomerulonephritis.

B-Vascular Diseases:

*-Acute Bilateral Renal Artery Obstruction As Caused Thrombosis And Or Embolism.

*-Disseminated Intravascular Coagulation (DIC).

*-Thrombotic Thrombocytopenic Purpura (TTP).

C-Interstitial And Tubular Diseases:

*-Acute Interstitial Nephritis Caused By Leptospira.

*-Acute Tubular Necrosis :This May Occur Toxic Or Ischaemic.

3-Post Renal Causes :

***-Acute Urinary Tract Obstruction Occur Due To :**

A-Stones In The Pelvis Or Ureters Or Urethera Or Urinary Bladder.

B-Prostatic Tumor Or Stressful Hypertrophy .

C-Blockage Of The Tubules By Casts Or Crystals Of Uric Acid Or Sulphonamides

Laboratory Findings :

1-Passage Of Small Amount Of Dark Urine (Oliguria).

2- Changes In Blood.

3- Hematological Changes .

1-Passage Of Small Amount Of Dark Urine (Oliguria)

A-Urine Contains Protein , Blood And Cellular Debris .

B-Specific Gravity Is Variable (Usually High)

2- Changes In Blood:

A-Retention And Increased Level Of Urea And Creatinine ,Potassium ,Magnesium ,Phosphate other Anions.

B-Metabolic Acidosis.

C-The Blood Calcium Is Some Lowered Secondarily To The Raised Blood Phosphate.

D-A considerable Increase In Extracellular Water.

E-Decrease In The Serum Concentration Of Sodium And Chloride.

3- Hematological Changes Include :

A-Rapidly Developing Anemia (Normocytic Normochromic Anemia) And Is Accentuated By Diluting Effect Of The Increased Blood Volume Secondarily To Water Retention .

B-Hemolytic Type Of Anemia Occurs To Due To Uremia.

C- Neutrophilic Leuckocytosis Is Also Present.

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THE TOPIC TODAY
CHRONIC RENAL FAILURE

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
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DEFINITION :

IT MEANS CHRONIC INSUFFICIENCY OF RENAL EXCRETORY AND REGULATING FUNCTIONS LEADING TO UREMIA

CHRONIC RENAL FAILURE RESULTS FROM PROGRESSIVE DECREASE IN GLOMERULAR FILTERATION AND THE LOSS OF TUBULAR FUNCTIONS.

CAUSES :

1-CHRONIC DESTRUCTIVE RENAL DUSEASES.

2-CHRONIC RENAL VASICULAR DISEASES.

3-OSBSTRUCTION TO THE URINE OUTFLOW.

4-CONGENITAL LESIONS .

5-METABOLIC CONDITIONS.

1-CHRONIC DESTRUCTIVE RENAL DISEASES INCLUDES:

1-CHRONIC PYELONEPHRITIS
2-CHRONIC GLOMERULONEPHRITIS
3-SYSTEMIC LUPUS ERTHEMATOSIS
4-BILATERAL RENAL TUBERCULOSIS
5-RADIATION NEPHRITIS

2-CHRONIC RENAL VASCULAR DISEASES :

1-MALIGENANT HYPER TENSION.
2-RENAL VEIN THROMBOSIS

3-OBSTERUCTION TO THE URINE OUTFLOW

1-THIS IS MANIFESTED BY BILATERAL HYDERONEPHROSIS AND OCCURS SECONDERILY TO URETHERAL OBSTERUCTION FOLLOWING :

1-ASTRICTURE

2-PROSTATIC ENLARGEMENT.

3-CARCINOMA OF THE CERVIX ,WHICH MAY ALSO OCCLUDE BOTH URETERS

4-CONGENITAL LESIONS AS :

1-POLY CYSTIC DISEASE
2-CERTAIN TUBULAR DEFECT MAY EVENTUALLY LEAD TO SEVER RENAL FAILURE

: - 5-METABOLIC CONDITIONS

1-HYPER CALCEMIA ,HYPOKALEMIA , AMYLOIDOSIS , GOUT
2-DIABETES MELLITUS (DIABETIC NEPHROPATHY)
ALL MAY LEADS TO SERIOUS RENAL DYSFUNCTION ESPECIALLY IF THEY PERSIST FOR LONG DURATION.

LABORATORY FINDINGS : INCLUDES :

1-DISTURBANCE IN WATER METABOLISM.

2-HYPONATREMIA.

3-MILD RETENTION OF THE POTASSIUM.

4-DISTURBANCES IN ACID BASE BALANCE .

5-DISTURBANCES IN CALCIUM AND PHOSPHORUS METABOLISM.

6-IMPAIRED GLUCOSE TOLERANCE AND INCREASED INSULIN RESISTANCE IS COMMON.

7-RETENTION OF WASTE PRODUCTS : UREA ,URIC ACIDS , CREATININE.

8-HYPERTENSION .

9-HEMATOLOGICAL EFFECTS

LABORATORY FINDINGS :

1-DISTURBANCE IN WATER METABOLISM : CLEARLY SHOWS :

***THE INABILITY OF THE FAILING KIDNEY TO CONCENTERATE THE URINE ADEQUATELY LEADS TO MODERATE POLYURIA ,WHICH IS CONSIDERED EARLY SIGNS IF RENAL FAILURE THE POLYURIA ALSO OCCURS DUE TO OSMOTIC OSMOTIC DIURESIS**

***POLYURIA IS ESPECIALLY MARKED IN CHRONIC PYELONEPHRITIS AS THE CONCENTRATING PART OF MEDULLA IS AFFECTED EARLY.**

***AMILD DEGREE OF DEHYDERATION OCCURS AND IS MANIFESTED BY THIRST.**

4-DISTURBANCES I ACIDBASE BALANCE :

PROGRESSIVE RENAL FAILURE IS ACCOMPANIED BY AMETABOLIC ACIDOSIS DUE TO RELATIVE FAILURE OF ACIDIFICATION OF URINE DUE TO IMPAIRENT IN AMMONIA PRODUCTION ,AND THE ABILITY TO SECRETE HYDROGEN ION (H+) IS IMPAIRED.

5-DISTURBANCES IN CALCIUM AND PHOSPHOROUS :METABOLISM

IN CHRONIC RENAL DISEASE:THERE IS APERSISTANT RETENTION OF PHOSPHATE AND SERUM PHOSPHATE RAISED

THE SERUM CALCIUM IS SOMEWHAT REDUCED AS AN INVERSE RESPONSE TO THE RAISED SERUM PHOSPHATE AND ALSO DUE TO IMPAIRED INTESTINAL ABSORPTION OF CALCIUM (BECOUSE OF VITAMINE D RESISTANCE AND DEFICIENCY)

SECOUNDARY PARATHYROID HYPERPLASIA MAY OCCUR ,THE PARA THORMONE MOBILIZES CALICIUM AND PHOSPHATE FROM THE BONES THEREFORE ,OSTEOMALACIA (RENAL RICKETS)OCCURRED.

9-HEMATOLOGICAL EFFECTS AND PATHOLOGICAL CHANGES:

SEVERE NORMOCYTIC NORMOCHROMIC ANEMIA IS COMMON IN THE LATER STAGES OF RENAL FAILURE . ANEMIA IS ATTRIBUTED TO THE IMPAIRED PRODUCTION OF ERYTHROPOITIEN,WHICH IS NORMALLY FORMED IN THE KIDNEY.

PURPURA ,WHICH IS PROBABLY DUE TO AQUALITATIVE DEFICIENCY IN THE PLATELETS.

أعوذ بالله من الشيطان الرجيم

بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

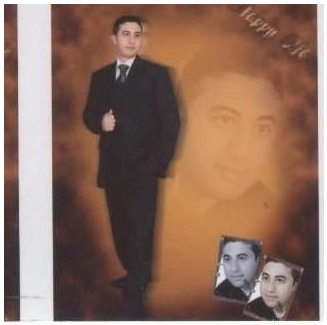
(ذلك من أنباء القرى نقصه عليك منها قائم وحصيد)

الأية ١٠٠ من سورة هود

بحث الدكتوراة وعليه ١٥ درجة فى التقييم العام ويتبع قسم الباثولوجيا الإكلينيكية للحصول على درجة الدكتوراة من جامعة بنها كلية الطب البيطرى بمدينة مشتهر محافظة القليوبية تم عرضه وإختياره وتنزيله من على شبكة المعلومات الدولية و ذلك لدراسة إمكانياته وتقيمه من ضمن الأجهزة الدقيقة فى تشخيص الأمراض المميتة الوبائية منها والوافدة وكيفية علاجها والحد من أخطارها.مقدمة من باحث مساعد

محمدمحمودمحمديوسف وهيبه .ماجستير البكتيريا والمناعة والفطريات بمعهد بحوث الصحة الحيوانية فرع طنطا التابع لمركز البحوث الزراعية التابع لوزارة الزراعة بجمهورية مصر العربية. للدراسة من قبل قسم الباثولوجيا الإكلينيكية .كلية الطب البيطرى بمشتهر.جامعة بنها العريقة.

رقم الجلوس الإختبارى : ٣٠١٤ المقرر : ٨١٤٢ .



جامعة بنها
شهادة مؤقتة
تشهد كلية الطب البيطري

شهادة
لائحة وشاحة

كشف طبي

شهادة تقدير

جمهورية مصر العربية
وزارة الصحة والسكان
كارت عائلة للتأمين الصحي الشامل

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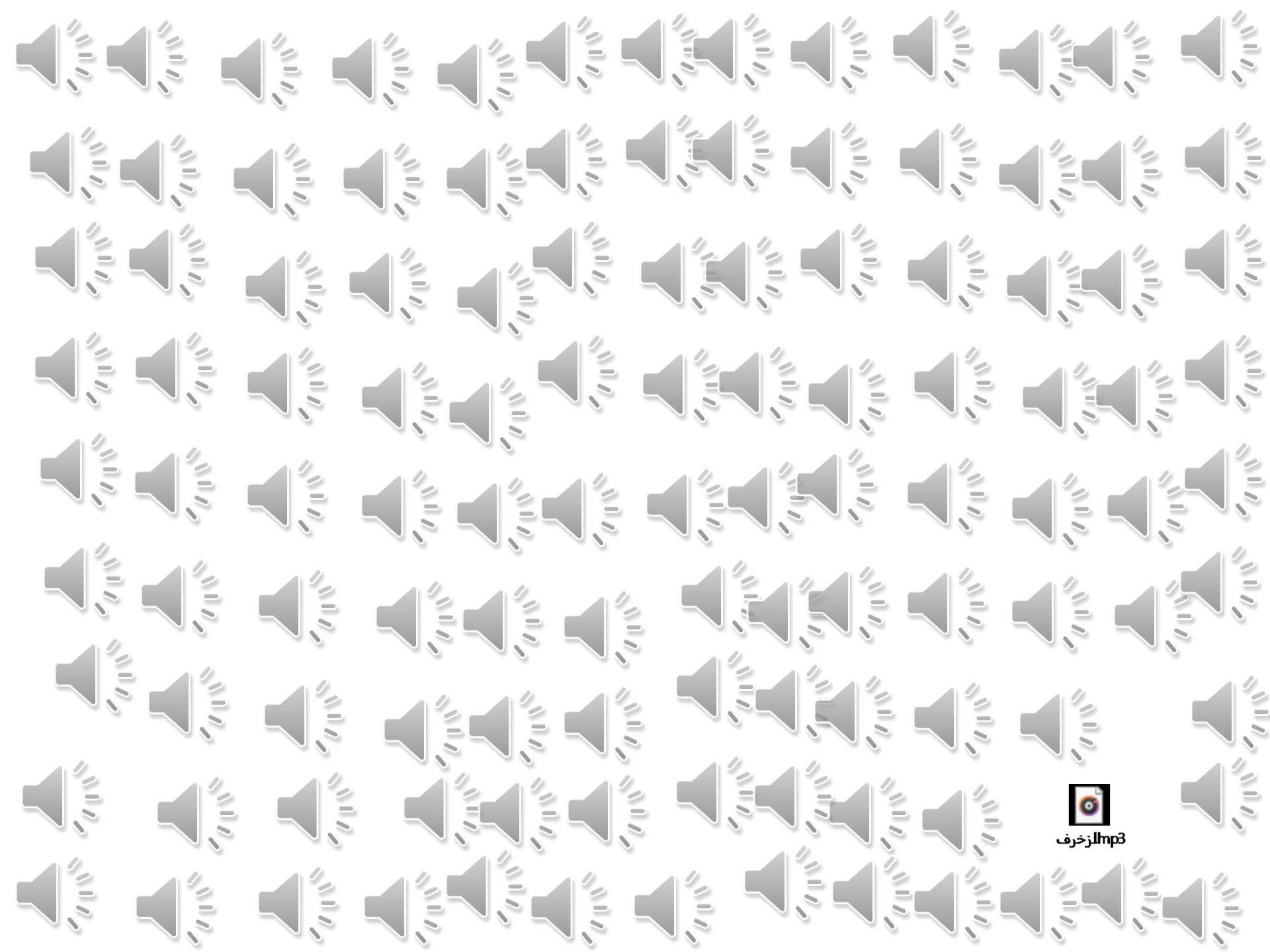
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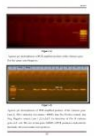

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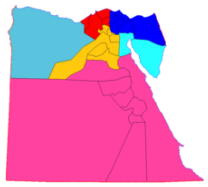

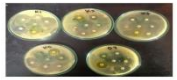

تلاوة خاشعة
MUSLIM HG

لو أنتم مؤسسة أو هيئة أو مقر أو شركة أو وزارة أو بناية أو جامعة أو كلية أو مدرسة أو معهد أو قطاع أو برج أو ناطحة سحاب أو منطقة أو جهاز أو إتحاد أو جمعية أو نوادى أو مديرية أو نظام أو تشغيل أو بيوت أوأركان أو دولة أو رئاسة أو محافظة أو بوابة أو حكومات أو لواءات أو فرق أو جيوش أو قارة أو أقسام أو غرف أو مبانى أو عمارات أومشيخة أو قاعات أو ندوات أو منظمات أو رواسيات أو مجمعات أو ميادين أودوائر أو لولبيات ولكم قيم ومنافع قصويات نرجوا معاليكم التدقيق لإيميلاتكم الإلكترونية الواقعيات والرسميات لتمحيص رسالاتى المرسلات لصناديق إيميلاتكم الإلكترونية لإحقاق الفوائد وتشرذيم الخسائر المصنفات مهلكات الثروة الحيوانية والطيور والدواجن المنبثقات كهرومغناطيسيات عبور من تلكم إيميلاتى وهى كالتالى :

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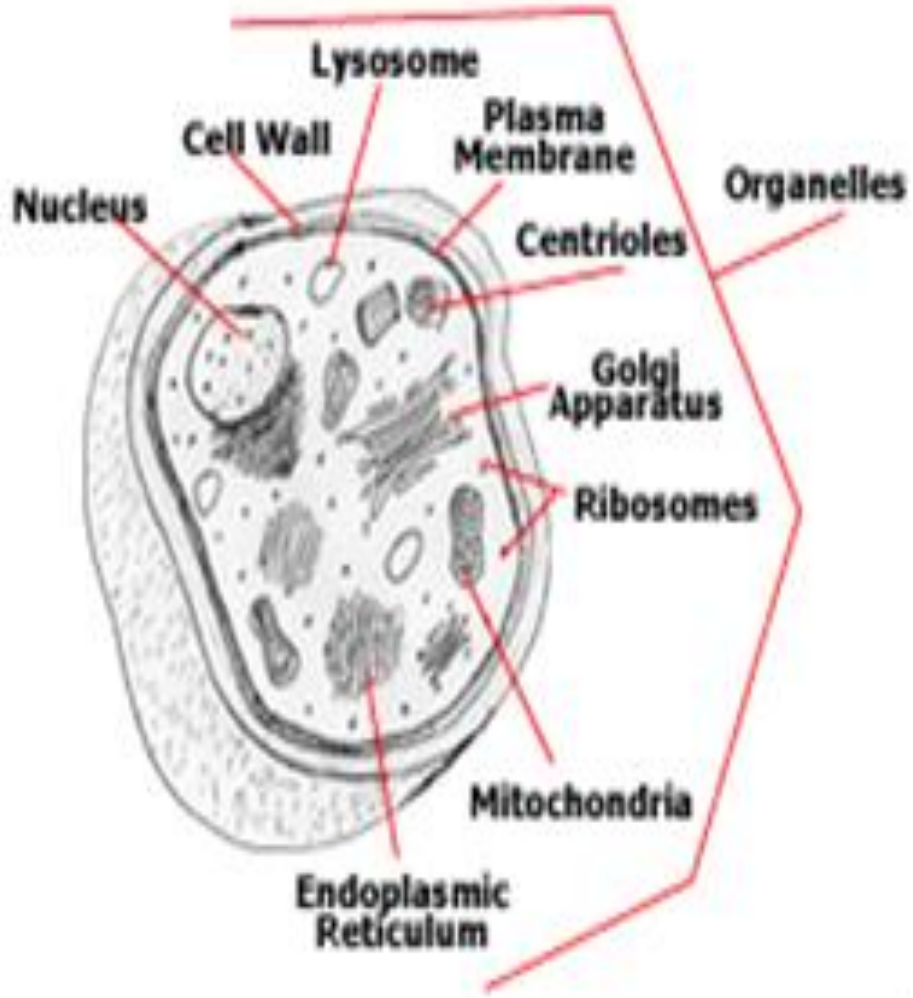
Mycology

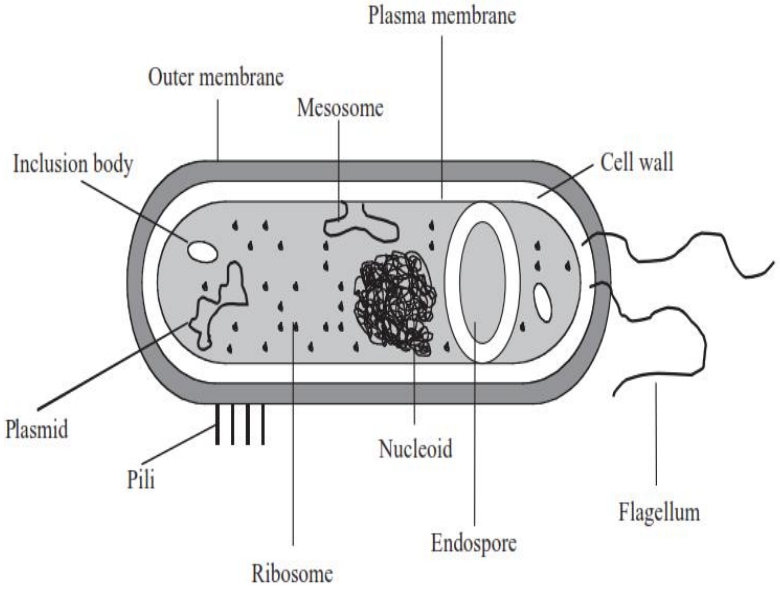
Mycology is science of studying Fungi**.**

- **Fungi** is eukaryotic cell or have true nucleus (the chromatin material surrounded with nuclear membrane).
- **Previously**, fungi were placed in division mycota of plant kingdom. Later on it placed in fungi kingdom.
- **Fungal cell differs** from eukaryotic cell in the structure of cell wall, cell membrane, nucleus and cell organelles.

Feature	Fungi	Bacteria
Nucleus	Eukaryotic	Prokaryotic
Diameter	Approximately 4µm (Candida)	Approximately 1µm (Staphylococci)
Cytoplasm	Contain cell organelles (Mitochondria, Golgi apparatus, ribosome and endoplasmic reticulum	Not Contain cell organelles
Cell membrane	Sterol present	Sterol absent except Mycoplasma
Cell wall content	Chitin	Peptidoglycan
Spores	Sexually and asexually spores for reproduction	Endospore for survival, not for reproduction
Thermal dimorphism	Yes (some)	No
	Spread radially by hyphae	

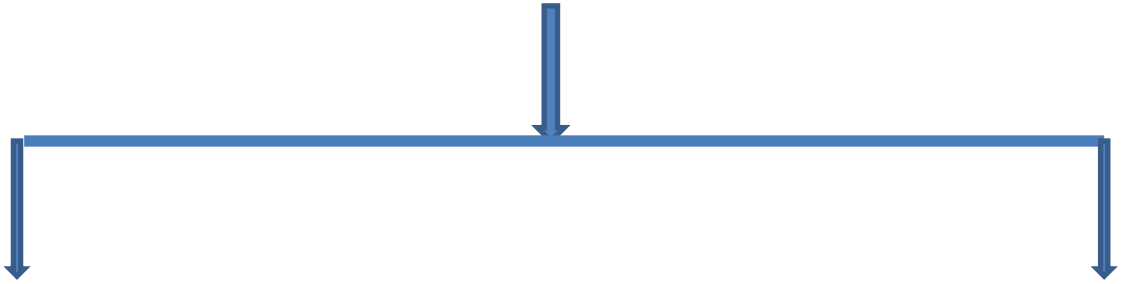


Fungal cell



Bacterial cell

Fungi grow as



Filaments or hyphae,

known as

Mould

Unicellular cells

known as

Yeast

Factors affecting fungal growth:

1. Nutrition

- All fungi are heterotrophs characterized by absorptive nutrition
- Growth of fungi depend up on:
  - organic substances as a source of carbon, e.g. sugars (glucose, sucrose, maltose,……..) some can use starch or cellulose.
  - Source of nitrogen as peptone, amino acids or amides, few of fungi can use nitrogen in inorganic combination as ammonium salts or nitrate.
  - Inorganic nutrition as potassium, phosphorus, magnesium and sulphur and only traces of iron, zinc, copper, magnesium and molybdenum.
  - Certain organic growth factors in very low concentration mainly vitamins e.g. thiamine.

2- Temperature:

- Most fungi are **mesophilic** (10-40 ^{o}C) with optimum temperature 25-35 ^{o}C and some fungi mainly pathogenic to 37 ^{o}C.
- Few fungi are **thermophilic or thermotolerant**, grow between 20-60 ^{o}C with optimum temperature 40 ^{o}C e.g. Asperigillus fumigatus.
- **Psychrophilic fungi** which grow at low temperature reach -20 ^{o}C e.g. Cladosporium, which cause proplems in frozen meat.

3- PH

- The optimum PH of fungi is range of 5-6.5 on the acid side.
- few fungi grow below PH 3.
- several acidophilic fungi can grow down PH 2 as many yeasts, species of Asperigillus, Penicillium and Fusarium.
- few basophilic fungi can grow between PH 10-11.

4- Oxygen supply:

- Aerobes: fungi need oxygen in at least small amount. The majority of fungi are aerobes.
- Facultative anaerobe: fungi which grow in absence or presence of oxygen.
- Obligate anaerobe: fungi which killed by short exposure to oxygen.

5- Water:

- Water is an absolute requirement for fungal growth.
- In the same time fungi are more resistant for water stress than other organisms.
- In drought, no growth occur but fruit bodies and vegetative resting phase capable of surviving.

6- Light:

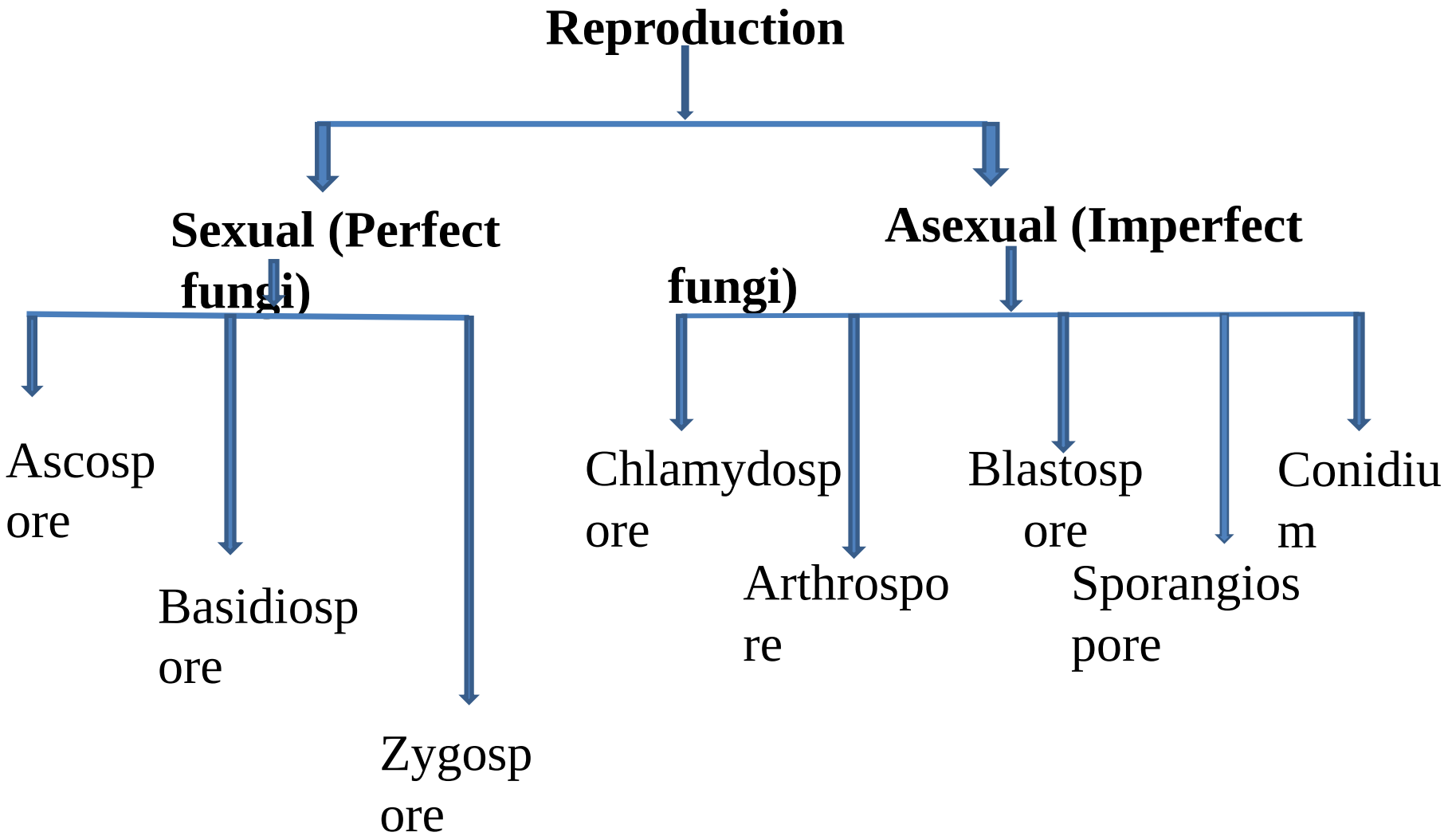
- Light have great role in reproduction since many moulds produce their conidia only in light.
- Vegetative growth is unaffected by light.

7- Radiation:

- UV radiation causes mutation and lethal damage for vegetative growth.

Reproduction of fungal cell

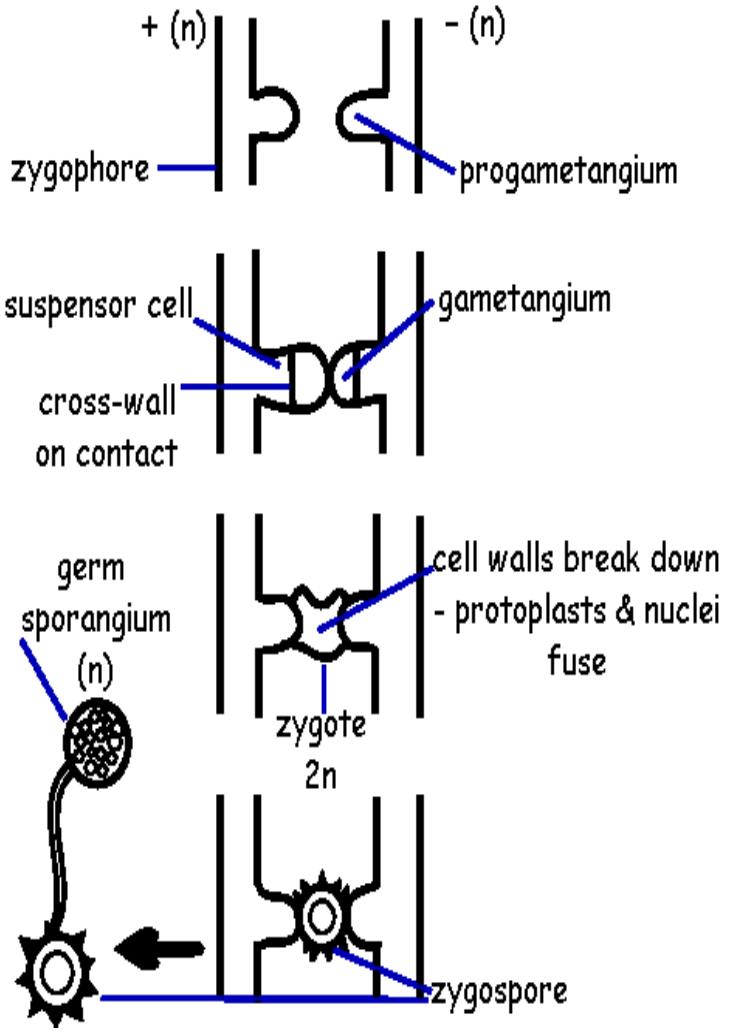
fungi reproduce by both sexual and asexual means



1- **Sexual reproduction:**

It consists typically of distinct phases;

- Production of sex organs and gametes.
- Fusion of gametes or sex organs (Plasmogamy).
- Nuclear fusion (Karyogamy).
- Meiosis followed by development of fruiting bodies and sexual spores.



Asexual (Imperfect fungi)

Chlamydospo
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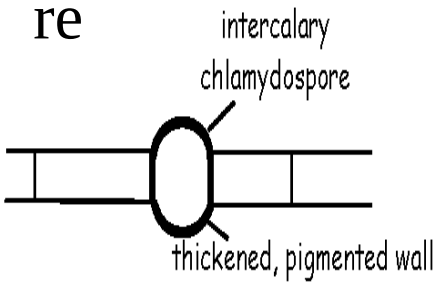

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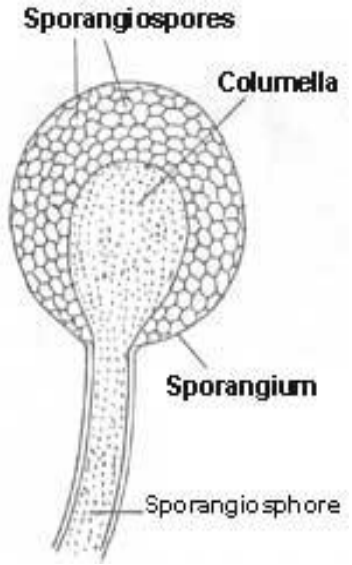

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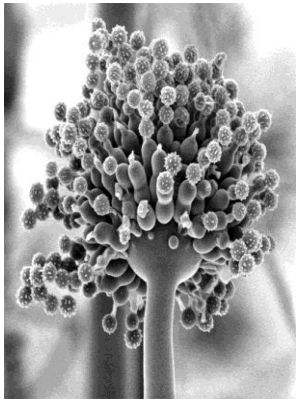
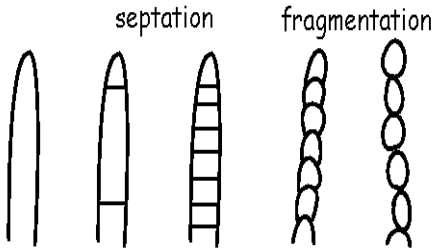


Arthrospore



Classification of fungi

1- Old Classification

Depending on hyphae and reproduction the kingdom of fungi contain five divisions:

	Chytridiomycota	Zygomycota or phycomycota	Ascomycota	Basidomycota	Deuteromycota
Hyphae	non septate hyphae	non septate hyphae	septate hyphae	septate hyphae	septate hyphae
Asexual reproduction	sac like known as sporangium produce motile zoospores	Sporangiospores produce non-motile spores	Conidia	Conidia	Conidia
Sexual reproduction	in some members, it is achieved	zygospore	Ascospore	Basidiospore	Absent or unknown

2- Recent classification

Fungi of medical importance can be divided into four major groups:

1. **Yeasts (Budding fungi)**

Unicellular round or oval cell reproduce by budding. They have no hyphae but some yeast may have unseparated budding cells that resemble hyphae. They are called pseudo-hyphae.

2- Moulds (filamentous fungi).

They grow in the form of filaments or hyphae. That may be septate hyphae or non-septate hyphae.

The part of hyphae grow under surface of agar and absorb nutrients, called vegetative hyphae. The rest of hyphae grow above the surface of agar and support reproductive structures they are called aerial

3- **Dimorphic fungi.**

They can occur in two different forms:

- Mould form when grow in nature or in culture at room temperature (25 ^{o}C).
- Yeast form when in infected tissues or when incubated at 37 ^{o}C.

4- **Dermatophytes**.

Dermatophytes are particular group of fungi that inhabit and are limited to most superficial parts of the body including keratin of stratum corneum and hairs causing an integumentary disease called ring worm or Dermatophytosis which characterized by circular areas of alopecia, scaling, crusts .

Dermatophytes

- **Dermatophytes**

are particular group of fungi that inhabit the most superficial parts of the body including keratin of stratum corneum and hairs causing an integumentary disease called ring worm or Dermatophytosis which characterized by circular areas of alopecia, scaling, crusts.

Classification of dermatophytes:

- **Zoophilic** dermatophytes are mainly found in animals but can be transmitted to humans.
- **Anthropophilic** dermatophytes are mainly found in humans and are very seldom transmitted to animals.
- **GeophiIic** dermatophytes are found mainly in soil, where they are associated with decomposing hair, feathers, hooves and other keratin sources. They infect both humans and animals.

- There are **3 genera** of dermatophytes
  - **Epidermophyton**
  - **Microsporum**
  - **Trichophyton**.

The most important species of dermatophytes:

A- Genus trichophyton:

1- *T. mentagrophytes*: Zoophilic, it is a frequent pathogen of rodents and dogs; occasional infections of cats, horses, cattle and man are observed.

2-*T. verrucosum*: Zoophilic cause the majority of ringworm infections In cattle and occasionally involves horses, dogs and other domestic animals.

3-*T. equinum*: zoophilic, frequently infects horses, human Infections occur occasionally and canine infection occur rarely.

4-*T. gallinae*: Zoophilic, infects poultry and infrequently other birds ard man. Infection in birds is limited to the skin usually the comb and wattles and does not invade the feathers, causes favus in poultry.

B- Genus Microsporum:

1- *Microsporum canis*: zoophilic, frequently infects cats, dogs and man, horses and lower primates are infected less frequently.

2- *Microsporum nanum*: Geophilic frequently causes mild infections in swine and infrequently involves man.

Reproduction

- **On the host** it reproduces asexually by formation of arthrospores.
- **On culture media** it reproduce asexually by formation of conidia which may be small in size (microconidia) or large in size (macroconidia). That differ in size and shape between dermatophyte species.

D

I. Clinical signs:

- Scaling or crusts on affected are

- Circular areas of alopecia (hair loss), as the hairs in the affected area are brittle and break near the skin surface, often giving the lesion a “shaved” appearance; truncated hair shafts may be seen through the scales and crusts.
- the lesion is circular in shape due to its central healing and spread infection toward the periphery of the lesion so it is called “ringworm”.

II. - Preliminary examination (wood’s lamp):

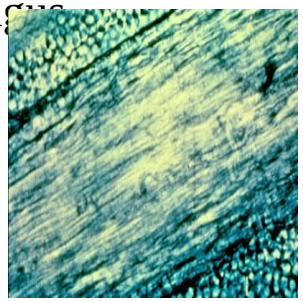
- Some dermatophytes strains, as *M. canis,* exhibit green fluorescence when stimulated by certain wavelengths of UV light.
- A Wood’s lamp can be used to examine the fur for these fungi.
- Certain topical preparations or alcohol may mask the fluorescence.

III- **Sampling**

- The samples are collected from periphery of the lesion including full thickness of keratinized epidermis until the blood is just drawn.
- The samples should be collected at paper envelope and submitted to the laboratory in inside additional warping, in dry condition.
- the site of the lesion should be washed firstly with water and detergent and allow to dry then wipe it with alcohol 70%.
- from periphery of the lesion collect skin scraping using blunt scalpel blade or microscopic slide.
- Damaged hair should be plucked from the lesion, never cut with scissors.

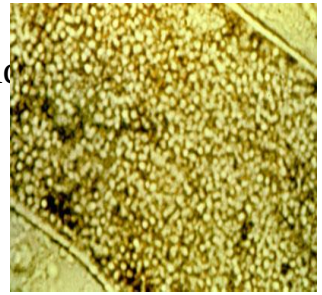
IV. Direct microscopic examination.

- On microscopic slide place the sample (hair or skin scraping) then add few drops of KOH 10-20 % to dissolve keratin layer which facilitate observation of the fungus.
- Then put cover slide and leave overnight.
- Examine under low and high power. Ectothrix
- The dermatophyte species appear as branching septate hyphae and chains of arthroconidia.



Ectothrix

- **In hair sample:**
  - Arthroconidia either accumulate outside the hair shaft) this indicate **ectothrix** arthroconidia which characteristic of all significant animal dermatophytes
  - or arranged inside hair shaft **(endothrix**) which common for anthrobophilic



Endoth

V. Culture characters:

- **Culture media used for isolation of dermatophytes:**
  - Sabouraud's dextrose agar with chloramphenicol (0.05 gm / litter) and cycloheximide (0.4 gm/litter).
  - *T. verrucosum* requires addition of thiamine and/or inositol for their growth.
  - *T. equinum* requires addition of nicotinic acid.
- Skin scrapings or plucked hair samples can be scattered over the surface of agar and gently pressed down on the medium with a swab or sterile forceps.
- Incubate aerobically at 25-30°c except *T. verrucosum* at 37°c for 3-4 weeks.

- Observe the observe and reverse colour of the colony, texture and growth rate.
- All these features are differ between dermatophyte species and it is diagnostic.

For example:

- ***T. Verrucosum*** is slow growing (3-4 weeks) and it has smooth, folded colonies. The observe side varies in colour from white or whitish grey and reverse side has white colour.

- ***T. equinum*** is Fast growing (10-14 d). Colony initially flat, white and fluffy but later velvety with central folding, cream observe colour and yellow to reddish brown reverse colou

- ***M. canis*** is fast growing (10-14 d). Its surface is white and cottony at the center with bright yellow periphery. Reverse side is bright yellow.

Microscopic examination of the colonies

- on the culture dermatophytes produce the reproductive structures (conidia) and hyphae which are characteristic for every genus and species.
- It can be observed under microscope by using a “slide culture technique” or by using adhesive side of clear cello-tape stripe and lactophenol cotton blue stain.
- The conidia may be macroconidia (multicellular) or micro-conidia (unicellular)

Slide Culture technique.

Used to observe conidia and hyphae of fungal colonies.

Procedures:

1. Using a sterile blade cut an agar block (1 x 1 cm) from a plate of Sabauroud's dextrose agar, small enough to fit under a cover-slip, and put it over a sterile glass slide.
2. Inoculate the four sides of the agar block with spores or mycelial fragments of the fungus to be grown.
3. Place a flamed cover-slip centrally upon the agar block.
4. Incubate the plate at humid chamber (put filter paper soaked with sterile dist in the peteri dish then put the slide over bended glass rod) at 26 ℃ until growth and sporulation have occurred.
5. Remove the cover slip from the agar block.
6. Apply a drop of 95% alcohol as a wetting agent.
7. Gently lower the cover-slip onto a small drop of Lactophenol cotton blue on a clean glass slide.

Differentiation between macro- and microconidia of dermatophyte genera

	Microsporum	Trichophyton	Epidermophyton
Presence	Usually present.	often absent.	present
Wall	Thick	Thin	moderately thick
Surface	Rough	Smooth	Rough
Shape (differ between species)	Ex. ***M.canis***: Spindle, mature spore end in distinct knob and of 6-15 cells.	***T.verrucosum***: Extremely rare, chains of chlamydospores are more characteristic.	***E. floccosum***: pear-shaped in clusters Septate into 2-5 cells.
Microconidia	Variable often absent if present they are tear shape and borne singly on hyphae.	Usually numerous and borne singly along hyphae or in grape like clusters.	absent

Yeast

- **Yeast is** unicellular spherical, ovoid, cylindrical or elongated cell.
- **Reproduction :**
  - **Sexually** some strains can reproduce by ascospore or by basidiospore.
  - **asexually** It reproduces by budding or fission.
- In some cases large number of cells sticks together after budding to form pseudo-hyphae or Pseudomycelia.
- **Pseudo-mycelia can differentiated from true mycelia by:**

1)True mycelia usually have refractive straight septa, while pseudomycelia has ends of daughter cells are curved and not retractive.

2) Terminal cells of true mycelium longer than the cells immediately preceding them, while in pseudomycelia the terminal cells is short or equal to adjacent cells.

Classification of yeasts

Three groups of yeast are recognized according to reproduction.

1- Ascomycetous yeast:

- Include yeast which has ascospore.
- The majority of them are not important as etiological agent of disease.

2- Basidiomycetous yeast:

- Characterized by formation of basidiospore.
- Not included as pathogenic yeast.

3- **Yeast imperfecti:**

- Included yeasts which not formed ascospores or basidiospores.
- The most important genera in this group are concerned with disease in man and animals are; **Candida**, **Cryptococcus**, Rhodotorula, Trichosporon.

Genus Candida

- It has more than 200 species from these large numbere only few incriminated as human and animal pathogen, for example; *C. albicans, C. guilliermondi, C. krusei* and *C. tropicalis.*
- *Candida spp.* are normal inhabitants of alimentary, upper respiratory, and genital mucosa of man and animals.

Genus Cryptococcus

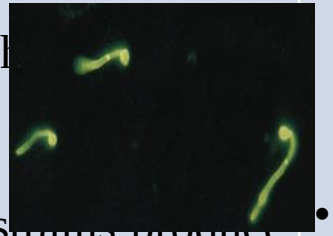
- It has about 17 species.
- *Cryptococcus neoformans* is the sole pathogenic species which cause cryptococcosis in man and animal.
- *Cryptococcus neoformans* has saprophytic nature and it isolated from soil enriched with pigeon excreta.
- *Cryptococcus neoformans* belongs to **Basidiomycetous yeast:**

Genus	Candida albicans	*Cryptococcus neoformans*
Speciemen	Skin scraping, vaginal discharge, sputum, milk, tissue.	Spinal fluid, sputum, pus, skin lesions, urine and milk of mastitic animals
Morphological characters		
shape	spherical or elongated	large spheroid, round or oval with thick wall
Chlamydospore	Present	Absent
Pseudo-hyphae	Present	Absent, it is appear as single or budding cell
Capsule	Absent	Present, detected by using diluted India ink 50%, which showing round cells surrounded by

Genus	**Candida albicans**	***Cryptococcus neoformans***
Culture characters		
Incubation temperature	25 - 30^{o} C	37 ^{o} C
Incubation period	2-3 days	3-5 days
Sensitivity to cycloheximide	Not sensitive	sensitive
On Sabouraud dextrose agar	With chloramphenicol and cycloheximide	With chloramphenicol only, Soft creamy opaque colonies in 3-5 days then become mucoid.
rice agar	Induce chlamydospore production and Pseudo-	-ve

Genus	**Candida albicans**	***Cryptococcus neoformans***
Biochemical test		
Sugar fermentation	+ve	-ve
Urea hydrolysis	-ve	+ve

Genus	Candida albicans	*Cryptococcus neoformans*
Specific diagnostic test	***Germ tube test:*** • Place several colonies from a non selective fungal medium in animal serum • incubates at 37 °C for 3 hours. • Microscopic examination reveals short hyphal segmentizing laterally from yeast cell, without constriction at the point of origin. the germ tube is half the width and 3-4 times the lengt cell. • Not all C. albicans strains produce germ tubes, so false negative may	**<u>Animal inoculation:</u>** • Intravenous injection of *Cryptococcus neoformans* in mice lead to death in 2 weeks with presence of capsulated yeast in spleen, lung and brain. • Intracerebrally injection of *Cryptococcus neoformans* in mice cause death in 4-14 days, before death the animal show hydrocephalus and circling movement. • Intranasal installation in mice produce primary pulmonary



Genus	Candida albicans	*Cryptococcus neoformans*
Pathogenesis: yeast infection is opportunistic, that made by lowering host defence by predisposing factors as: • Antibiotic therapy • Immunosuppressive disease • Glucocorticoid therapy	➢ *C. albicans* cause of superficial and systemic candidiasis. ➢ Cutaneous candidiasis and otitis externa occurs in dogs and cats. Cutaneous candidiasis associated with dermatitis the muzzle, inguinal area, scrotum, and dorsal and lateral aspect of the feet. ➢ In calves and foals *C. albicans* cause gastroesophageal candidiasis associated with gastric ulceration. Animals present clinically with signs of colic and watery diarrhoea that are non responsive to medical treatment. ➢ In poultry , it causes crop mycosis (thrush). The crop, mouth, oesophagus,	➢ it cause Cryptococcosis is an acute, sub acute or chronic mycosis that can be pulmonary, systemic or meningeal. ➢ In horse, it causes nasal granuloma and meningoencephalitis. ➢ In cattle, it cause mastitis, nasal or pulmonary and CNS affection. ➢ In sheep and goat it cause septicaemia with pulmonary and meningoencephalitis.

Order: EUBACTERIALIS

Family: BACILLACAEA

Genus: BACILLUS

Bacillus Anthracis

These organisms are aerobic, spore bearing bacilli which are cylindrical, flagellated, grow in chains and produce large irregular colonies on solid media .liquefy gelatin.
Only on e species *Bacillus anthracis* is pathogenic
They are found in the soil and on decaying vegetation where they are most active in bringing about the decomposition of organic substances .because of the resistance of the spores to high temperatures and desiccation .these organisms are among those which most frequently contaminate culture media in the laboratory as anthracoids.

Definition:

BACILLUS ANTHRACIS:

It causes anthrax all over the world. The organism spread by the soil contaminated feeds, and by water, pasture lands remain contaminated for years by the spores of the organism. Carnivorous animals as dogs, cats and hogs contract anthrax by feeding on dead carcasses.

Man contracts the disease by contact with infected animals, by handling infected hides, by sorting wool, from shaving brushes and from insect bits

Morphology and staining:

The bacilli are cylindrical rods, (1 to 1.2 µ) in diameter by (3-8µ) in length. The capsule is formed in tissues of the infected animal and is demonstrated by polychrome methylene blue stain. The spores are ellipsoidal situates centrally in the cell and are (0.7 µ – 0.8 µ × 1.5µ) in size, the spores are not formed in the animal body .the organism is gra .

Cultural characters:

Bacillus anthracis grows readily on blood agar and isolated easily from the food or internal organs of infected animals.
It is aerobic; grow well at 37ºc and p

On nutrient agar, surface colonies appear dull, opaque , grayish-white with an irregular border from which long strands of cells are seen in parallel arrangement the typical "medusa head" characteristic appearance.

In broth, is turbid with a floccular growth on the surface which sinks to the bottom of the tube within 24 hours.

In a gelatin stab culture, filaments of growth radiate from the line of puncture and give the appearance of inverted fire tree.

The organism losses its capsule when grown on artificial media unless the medium is incubated in an atmosphere of 10% co2.
Contrary to the usual type of variation, rough (R) is normal and virulent whereas smooth (S) colonies are avirulant.

Resistance and viability:

The vegetation cells of *Bacillus anthracis* are not more resistant

Spores are resistant and remain viable for many years in the soil.

The organism is rapidly destroyed by decomposition in un opend carcasses after the third day.

10 % formalin at 40 ºc will kill anthrax spores in 15 minute.

Freshly prepared 5% sodium hydroxide is satisfactory agent for disinfecting objects contaminated by the spores of this organism.

Biochemical properties:

Bacillus anthracis forms acid but not gas from glucose, sucrose, maltose, fructose and dextrin.
Some strains produce slight acid in glycerol amd salicin .
The organism not ferments lactose, galactose and mannitol.
Indol and H2S are negative while methylene blue is reduced and MR positive and VP negative.
Litmus milk is coagulated, decolorized and slowly peptonized.

Antigenic structure and toxins:

Bacillus anthracis has two antigenic substances (capsular antigen and somatic polysaccharides antigen).both antigens act specifically with precipitating sera. No antigenic groups have been reported for the organism.

Bacillus anthracis produces two toxic substances when grown in acasamino acid medium without serum or other proteins. One component called protective antigen and the other called **filter factor**, the protective antigen migrates in an invisible component intermediate between the gamma and beta globulins it is destroyed by heating at 57 ºc for 30 minute and by trypsin .

Pathogenicity :

Bacillus anthracis is pathogenic for cattle, sheep, horses,mules,swine,dogs and cats. It causes septicemia in the first four species of animals.

On P.M, anthrax is characterized by oedematous infilterations in the subcutaneous, haemorrhages in various parts of the body especially in the serous membranes, marked swelling of the spleen, which is dark red and soft, the blood is dark red, tarry and does not clot. Hemorrahages are often observed from the natural body openings.

Cutenous anthrax :

Resulted from skin infection through scratches or other breaks in the skin. Regional lymph nodes are swollen and painful.

Intestinal anthrax:

Resulted from the eating of uncooked meat from an anthrax carcass and is extremely rare in civilized countries.

Respiratory anthrax:

It is acquired through inhalation of the air when working with materials contaminated with bacillary spores.

Immunity:

Two vaccines were used:

Pasteur vaccine 1: attenuated by growing at 42ºc for 12 day , it kills mice but not guinea pig or rabbits.

Pasteur vaccine 2: attenuated for shorter time, about (5-7 days) so it kills mice and guinea pigs but not rabbits and sheep. By using pasteur vaccine 2 simultaneously with immune serum.

By using ***spore vaccine*** which is attenuated to correspond to pasteur vaccine 2 and it is given intradermal.

Serum therapy in anthrax has been demonstrated to be dependable.

Diagnosis of anthrax:

In submitting material for anthrax diagnosis; great caution must be exercised, it is an unnecessary risk to autopsy carcass which has obviously died of anthrax, sending an ear packed in a tight suitable container is universal practice, also sending of blood smears, cotton swabs, and small vial of blood or tissues is preferable if sufficient caution is taken.

By Ascoli´s test:

It depends on the occurrence of specific precipitation in the serum of an immunized animal.
Serum with known precipitating properties must be selected.
An extract of the tissues is made in slightly acidified saline.
The fluid is boiled for extraction, then filtrate through filter paper.
0.5ml of serum is placed in a narrow tube and the filtrate is carefully run onto the top.
The development of white ring of precipitate at the junction of the two fluids denotes positive result.

Characters	*B. anthracis*	*B. anthracoids*
Motility	Non motile	Motile
Capsule	Present	Abscent
Shape and position of spore	Oval, cenrta,not bluged	Vary in shape without bluging
Chain formation	Growth in long chain	Singly,diplo,short chain
Growth in broth	Cotton wool fluffy deposit	Turbidity or pellicle
Growth in galatin stab	Inverted fir tree	Fire tree absent
Haemolysis	Weak or no	Strong (β)
Pathogenicity for : **Mice.** **G.P** **Rabbit.**	 Die (24 hr.) Die (48 hr.) Die (72 hr.)	 Large dose are pathogenic. Non pathogenic. Non pathogenic.

Genus : Brucella

- Species :
- Br.abortus
- Br.melitensis
- Br.suis
- Br.ovis
- Br.canis
- Br.neotoma

Genus : Brucella

- Species :
- Br.abortus
- Br.melitensis
- Br.suis
- Br.ovis
- Br.canis
- Br.neotoma

Morphology & staining

- **Gram –ve**
- **Acid fast coccobacilli**
- **Singly arranged**
- **In old culture short chains**
- **Motile**
- **Non capsulated**
- **Non sporulated**
- **In tissues, occurs in groups & it is intracellular**

staining

- **Modified Zeil –Neelsen stain**

decolorization with 0.5% acetic acid

- **Koster´s stain**

depends on : brucella is alkaline fast

brucella appear as orange red against blue background

Cultural characters

- **Aerobic M.O**
- **Br.abortus need 5-10% CO2 for primary isolation**
- **Special media are needed with antibiotic inhibitory substances as:**
- **- albimi agar with ethyl violet**
- **- tryptose media with actidione**
- **-brucella agar with polymyxin**
- **-liver infusion serum agar**

- **Requires 3-4 days incubation**
- **Colonies after 2-3 days small 0.5 mm ,round,convex, translucent with small glistening surface (smooth colonies)**

- **Rough colonies are flatter, large with granular appearance & pale yellow .**

- **In tryptose broth :**

  turbidity develops slowly + fine granular deposit which become viscous after prolonged incubation

Biochemical characters

species	oxidase	H2S production	Nitrate reduction	Urea hydrolysis
Br.abortus	**+**	**+**	**+**	**+**
Br.melitensis	**+**	**Neg**	**+**	**+**
Br.suis	**+**	**Neg**	**+**	**+**
Br.ovis	**Neg**	**Neg**	**Neg**	**Neg**
Br.neotoma	**Neg.**	**+**	**+**	**+**
Br.canis	**+**	**Neg**	**+**	**Neg**

Antigenic structure

- **Smooth strains posses 2 antigens**
  A & M
  A:M ratio (quentative difference)

By aggl.absorption test using monospecific A or M antisera

Used with other tests to differentiate 3 main species:

Br.abortus **20 : 1**

Br.melitenesis **1 :20**

Br.suis **1 : 1**

Symptoms & lesions

- **Human → milk ,meat ,contact or M.M**

  malta fever, undulant fever or mediternian fever

Cows → contagious abortion or Bang´s

disease abortion 7-8 month of pregnancy

virulent strains have a protective outer protein coat allowing them to survive intracellular → chronic infection

- **Bulls** can be isolated from L.nodes of the digestive tract & spleen
- No lesions recognized

- **Sheep & goats**

Br.melitenesis

- **abortion 3-4 month** of pregnancy .
- lameness & mastitis
- The udders become infected in a high percentage of cases
- **Br.meletenesis is shed in milk**

- **In bulls → epididymitis & testicles abscesses usually develops & the organs are destroyed**

Laboratory diagnosis

- 1- history 2-samples

source	types	examinations
Aborted fetus	Stomach content Intestinal content Lung tissue. spleen	Direct smear bacteriology
Mother after abortion	Uterine discharge Fetal fluid	Direct smears, bacteriology G-pig inoculation
2-3 weeks after abortion	Blood Milk whey	Serological : Abortus bang test Tube aggl.test
bulls	Preputial wash Semen serum	G,pig inoculation Bacteriology Serological test

- **3- direct smears:**

Fresh samples of chronic tissues

Fetal stomach content

Uterine discharge

Films stained by the differential stains for brucella

Brucella occurs characteristically in clumps intracellular as well as extracellular

Bacteriological examination

- **Isolation of brucella**

On the special media containing antibiotic

Incubated 37ºCfor 10 days at least

Br.abortus & melitenesis required 5-10% CO2

For primary isolation

- **Differentiation of the isolates**

tests	Br.abortus	Br.melitenesis	Br.suis
CO2 requirment	+	-	-
H2S production	+	-	-
Urease hydrolysis	+ (1-2 hr)	variable	+ (0-5 hr)

Characters	*Br. abortus*	*Br. meletensis*	*Br. suis*
Dye sensitivity:			
Basic fuchsin	+	+	-
Methyl violet	+	+	-
Thionin	-	+	+
Pyronin	+	+	-
Antigenicity (A:M ratio)	20:1	1:20	1:1
Agglutination by:			
Monospecific M antisera	+	+++	++
Monospecific A antisera	+++	+	++
Susceptible animals	Cattle	Sheep and goat	Swine
Virulence to man	Weak	High	moderate

- **G.pigs inoculation :**

The infected material inoculated S/C in 2 male G.pigs to obtain a pure culture of brucella

+ve → orchitis i,e strauss reaction within 3-8 weeks

The organism in pure culture can isolated from spleen ,liver & testicle

The serum of slaughtered G.pigs can be examined the presence of antibodies

Serological tests

- **For brucella diagnosis by indirect detection of antibodies in serum , vaginal mucous, milk, whey & semen –serum**

- **1-agglutination test :**
- **1.1 : serum aggl. test or tube aggl. test (SAT) :**
- **By using standard Br.abortus**
- **+ve titer = 1/40**
- **Suspected titer =1/20**
- **Accurate, simple**
- **The test repeated after one month**

- **1.2 : plate rapid method or slide agglutination test :**
- **Stained heavy suspension of Br. abortus killed antigen**
- **Applied to give screening on a herd level**
- **Serum ,whole blood . Whey may be used**
- **The result read in several min.**

- **1.2.1 : Rose Bengal test (RBT) :**

Is a type of slide agglutination

by using stained antigen with Rose-Bengal

- **The test is rapid & it give –ve reaction**

- **used to differentiate between vaccinated from infected animals**

- **2- Abortus Bang Ring test (ABRT)**
- **Or Milk Ring Test (MRT):**

1 drop of stained (heamatoxylin , blue or tetrazolium ,red) heavy suspension of killed Br.abortus + 1 ml of milk

Or 1 ml antigen + 9 l milk then mixed well

is incubated at 37ºC in water bath for 1-2 hr especially for buffaloes , sheep, goat milk

-ve	+ve
No change	decolourized milk
Bluish color	capped by bluish violet cream

Used extensively in herd level for survey purposes & general screening method

- **3. complement fixation :**

Used for detection of chronic infected animals

In case of repeated aggl. Tests give unreliable results

4. Fluorescent antibody technique:

Used for examining the highly contaminated materials such as :

retained placenta , cotyledons or vaginal discharge

Immunity & control

- **The immunity is depending upon cell mediated immunity by using attenuated vaccines**
- **The commonly used vaccines are:**

1- buck strain :

Avirulent strain of brucella to cattle

It is administered as a single S/C dose

Vaccination of calves (3-12 months) as widely used as calf hood vaccination

- **2- McEwen 45/20 vaccine (rough brucella abortus vaccine) :**

- **It is not virulent enough to cause disease in cattle**

- **The rough 45/20 does not produce agglutinins in animals (advantage over than strain 19)**

- **3- a killed 45/30 adjuvant vaccine (K45/20A):**

To avoid the disadvantages of the rough strains

Order: EUBACTERIALIS

Family: LACTOBACTERIACE

Genus: STEROTOCOCCUS

SPECIES:

Sterptococcus pyogenes

Sterptococcus agalactiae

Sterptococcus dysagalactiae

Sterptococcus uberis

Sterptococcus equi

Sterptococcus zooepidemicus

Sterptococcus canis

Sterptococcus gallinerum

General characters of Streptococci:

- It is spherical or ovoid cocci in shape 0.5 µ in diameter, it is usually arranged in chains of various length, gram positive .they are non motile, non sporulated but some strains are capsulated (slime layer).

- **<u>Growth requirements and cultural characters:</u>**
- Streptococci are aerobic or facultative anaerobic, grow weak on ordinary media but grow well on blood or serum agar at 37ºc and ph 7,2 . The organism produces haemolysis on blood agar plate the specific media are ***Edward´s blood ager*** media and sodium azide agar media.
- Streptococcus doesn't produce pigments either in fluid media or on solid media. The colonies on blood agar are small, smooth – glistening after 48 hours, while in broth granular growth and powdery sediment are formed within 36-48 hours.

- **<u>Classification:</u>**
- **1-according to type of haemolysis :**
- Streptococci causing complete haemolysis (Beta (β) haemolysis), a clear zone of haemolysis as *Strept.pyogenes* and *Strept.equi*.
- Streptococci causing in complete haemolysis (alpha α) such as *Strept.viridians* and *pneumococci*.
- C) Streptococci causing no haemolysis (gamma γ), non hemolytic streptococci as *Strept.lactis* and *Sterpt.salivaris*.

- **2-Lancifield classification:**
- Lancefield reported that the hemolytic streptococci could be divided into groups (A-N) on serological basis depending on precipitation test using the specific carbohydrate substance designated as C.substance as the antigen which differed in each group:
- - Group A: *Streptococcus pyogenes*
- - Group B: *Streptococcus agalactia*
- -Group C: animal streptococci as *streptococcus zooepidemicus*, *Streptococcus dysgalactia, Streptococcus equi, Streptococcus canis.*
- -Group D: Enterococci as streptococcus fecalis ,*Streptococcus faecium*
- -Group E: Strains isolated from milk and dairy products as *Streptococcus uberis*
- -Group F: Strains isolated from respiratory tract.
- -Group G: Strain isolated from respiratory tract of man and dogs.
- -Group H, K, M and group N

- **3-Sharman classification:**
- It depends on the biochemical characters of the enterococci or non pathogenic streptococci on the following reactions:
- Growth at 10 ºc and 45ºc as *Strept.viridians*.
- Tolerance to 6.5 % sodium chloride.
- Reduction of methylene blue.

- ***Streptococcus pyogenes***
- **Morophological characters:**
- *Strept.pyogenes* are arranged in long chain, it is cocci , non motile , non sporulated, has capsule in young culture and gram positive
- **Distribution:**
- It is widely distributed on the skin and in the mucous membrane

- **Growth requirements and cultural characters**:
- *Strept.pyogenes* grow on media containing blood or serum at 37ºc and PH 7.2 .
- Its colonies on the solid media are very small, smooth glistening, dew drop like, finally granular and not pigmented.
- The colonies produce zone of complete haemolysis (beta haemolysis)on blood agar after (24-48) incubation

- **Biochemical properties:**
- The organism ferments glucose, lactose,sucrose, and salicin producing acid only do not ferment sorbitol, do not liquefy gelatin, catalase negative and indol negative.
- **Resistance:**
- *Strept. pyogenes* killed by 60ºc for half hour , so laboratory cultures should be stored at (3-5ºc) in blood broth .
- The organism is sensitive to sulfonamides and penicillin.

- **Streptococcus toxins and enzymes:**
- It produces two soluble haemolysins which cause haemolysis of erythrocytes.
- *Strept. pyogenes* produces a leucocyte destroying substance known as leucocidine.
- Itproduces erythrogenic toxin which is responsible for the characteristic skin lesions of scarlet fever.
- Produce fibrinolysin ,proteinase, hyalouronidase (spreading factor).

- **Antigenic structure**
- Somatic (o)antigen
- Capsular antigen
- (m) protein only in s.pyogen
- **<u>Pathogenicity</u>**:
- *Strept. pyogenes* causes a number of disease in man.
- It is associated with scarlet fever, septic sore throat, tonsillitis and various suppurative inflammatory conditions.
- The organism invades the bovine mammary gland producing mastitis.
- Some strains of *Strept. pyogenes*are highly virulent for the mouse and rabbit, guinea pig is more resistant.
-

- **Laboratory diagnosis of *Strept.pyogenes* infections:**
- By direct smears of the exudates from the lesions of the disease usually reveal long chains of streptococci.
- Haemolysis of blood media.
- Lancifield technique with various biochemical tests.

- =**Differences between *Staph.aureus* and *Strept. pyogenes*:**

Characters	*Staph. Aureus*	*Strept. Pyogenes*
Arrangement	Grape like clusters	Long chains
Growth	After 24 hours	After 48 hours
Turbidity in broth	Turbid and thick sediment	Clear and granular sediment
Colonies on ordinary media	Round, convex, opaque and pigmented	Minute, transparent, non pigmented
Catalase test	+	–
Gelatin liquefaction	+	–
Methylene blue	+	–
Bactiracin	resist	Sensitive

- **MASTITIS STREPTOCOCCI:**
-
- ***Streptococcus agalactiae•***
- ***Streptococcus dysgalactiae•***
- ***Streptococcus uberis•***
- These three species of streptococcus are biochemically and serologically different; however, they are associated with bovine mastitis.

- **Morphology and staining:**

- Gram positive cocci
- Arranged in long chain(s. agalactica, s. disagalactiva) short chain (s. ubris)
- Non motile, non sporulated, non capsulated)

- **<u>Growth requirement and cultural characters:</u>**
- These organisms grow well on blood or serum agar, also the use of sodium azid crystal violet blood agar facilitates the isolation of these organisms.
- It is aerobic and microaerophilic and grow at 37 ºc.
-
- *Strept. agalactiae* and *strept. dysgalactia* usually produces flocculent growth in the bottom of the tube with clear supernatant liquid above while *Strept. ubris* produce a uniform turbidity in both medium.
- *Strept. agalactia* produces beta haemolysis on agar if it is refrigerated after 24 hours incubation at 37ºc while *Strept. dysgalactia*and *Strept. uberis*are non haemolytic

- **Biochemical reaction:**
- Indol negative, doesn't liquefy gelatin and doesn't reduce nitrate.
- *Strept. agalactiae* and *Strept. dysgalactia* do not ferment sorbitol while *Strept. ubris* ferment sorbitol.
- *Strept. agalactiae* ferment salicin while *Strept. dysgalactia* do not ferment salicin.
- Esculin hydrolysis in *Strept. ubris* is positive

- **<u>Resistance and viability:</u>**
- As *Streptococcus pyogenes*.
- **Antigenic structure and toxin:**

Somatic antigen only

Toxins

Haemolycin , leucocidin

- **<u>Pathogenicty :</u>**
- Three organismscausepranchymatousmastitis in cow, sheep and goats

- **<u>Laboratory Diagnosis:</u>**

- Isolation and identification of streptococci from milk samples.
- By **Hottis and miller** test for the detection of *Strept. Agalactiae*, this test is performed by adding 0.5 ml of sterile 0.5 % aqueous solution bromocresol purple to 9.5 ml milk. The milk is incubated 24 hours at 37ºc and the test is read where the appearance of canary-yellow colonies of bacterial growth along the walls and in the bottom of the test tube is diagnostic of *Strept. agalactiae* infection .

Differentiation between the 3species of streptoccoci caising mastitis:

Test	*St.agalactiae*	*St.dysgalactiae*	*St.ubris*
1)lancifield group	B	C	E
2)haemolysis on blood		γ(α)	γ
3) Growth on bile salt.	Well		
4)CAMP test	+		
5)Na hippurate test	+		+
4)Aesculin hydrolysis.	–		+
7) Type of mastitis.	Chronic	Acute	Subacute

STREPTOCOCCUS EQUI

- As s. pyogen

Pathogenesis

Cause strangle

	strangle	glander
	In upper respiratory tract Cause respiratory manifestation	In upper respiratory tract Cause respiratory manifestation
Age of animal affected	More than 5 years	Less than 5 years
Causitive agent	s. equi	Pseudomonus mallie

Family : pasteurellaceae

- Gram negative
- Short rods
- Non sporulated
- Facultative anaerobes
- Fermentative metabolism
- Some of them parasitic to the mucous membranes of mammals & birds

Famiy Pasteurellaceae is differentiated into :

1- Genus : Pasteurella

2- Genus : Haemophilus

3- Genus : Actinobacillus

Genus : Pasteurella

- Characterized by **the bipolarity** in films from infected tissues

- Parasitic in respiratory tract

Family : Pasteurellaceae

Genus : Pasteurella

Species : P.multocida

Genus : Pasteurella

- **General characters :**

- Gram –ve, short coccobacilli
- Non motile
- Facultative anaerobic
- Catalase & oxidase positive
- Fermentation of CHO produce acid ,no gas
- Bipolar staining, especially in organ smears
- The **Pasteurellae** are frequently pleomorphic & form long chains in passage cultures
- Has 11 different species

p.multocida

- Morphology :
- Gram –ve , ovoid to short rods coccobacilli
- 0.3-1x 1.3 µ
- Arranged singly or short chains
- Bipolar character by leishman´ stain
- Non motile
- Non sporulated
- Most of the virulent strains have slime layer (complex polysaccharide) mucoid or smooth

Cultural characters

- Facultative anaerobes
- Grow well in presence of blood or serum after 24 hours at 37ºC
- **In serum broth glucose broth**

  slight turbidity after 24-48 hrs

  + sediment
- Onto **sheep blood agar** from fresh materials

  after 24-36 hrs in 3 forms :

- Onto sheep blood agar from fresh materials after 24-36 hrs in3 forms :

Mucoid colonies	Smooth colonies	Rough colonies (blue variant)
moist,Round,convex,sticky Large 2-5mm Mucoid in consistancy With large slime layer of hyaluronic acid Highly virulent strains For rabbits & pigeons	round, grayish,1mm glistening ,smooth Possesses small amount of slime layer Moderate virulence for Mice & rabbits	isolated mainly from chronic cases after subcultures Show auto-agglutination why ????????? Virulent to rabbit

- **DAS media**
- **Morres media**

selective and specific media for isolation of Pasteurella

Biochemical reactions

- Catalase +ve
- Oxidase +ve
- It is essential to differentiate between

species	lactose	mannitol	B haemolysis	MacConkey	Indole	H2S	urea
P.multocida	-	+	-	-	+	-	-
P.haemolytica	+	+	+	+	-	-	-
P.gallinarum	-	-	-	-	-	-	-
P.aerogenes	-	-	-	+	-	-	+

Antigenic structure

- **Somatic O-antigen** : 1 -11 types

- **Capsular K-antigen** (surface antigen): according to **Carter** to 5 types A,B,C,D,E

According to **Roberts** to 4 immunological variants I,II,III,IV in mice

-

K-antigen	O-antigen	serotypes
A	1,3,5,7,8,9	1:A
		3:A
B	6,11	6:B
		11:B
D	1,2,3,4,10	1:D
		2:D
E	6	6:E

Epidemiology & pathogenesis

- Affect all animals& birds

  septicemia,acute respiratory diseases,with sudden death
- **Mode of infection :**

-normal inhabitant of upper respiratory tract

Due to :

-weather

-long shipping

-faulty nutrition

-Secondary infection

Diseases caused by p.multocida

- **Cattle & buffaloes**

haemorrhagic septicemia

Sheep

pleuropnumonia

mastitis

Horse

contagious pleuropnumonia

Rabbits

snuffle

chronic abscess

Fowls

fowl cholera

Laboratory diagnosis

- History
- Symptoms & P.M lesions
- Samples

- In septicemic cases : all paranchymatous organs
- - in purified caracasses : in bone marrow
- -in chronic cases : in the affected lesion

- Staining with leishmans stain:

- From blood or tissues showed bipolarity (characteristic feature of pasterurella

- **Isolation: on selective media as DAS & Morres media**
- **Differentiated biochemically**
- **Demonstration of the pathogenicity of the isolates by ????**
- **Differential diagnosis :**

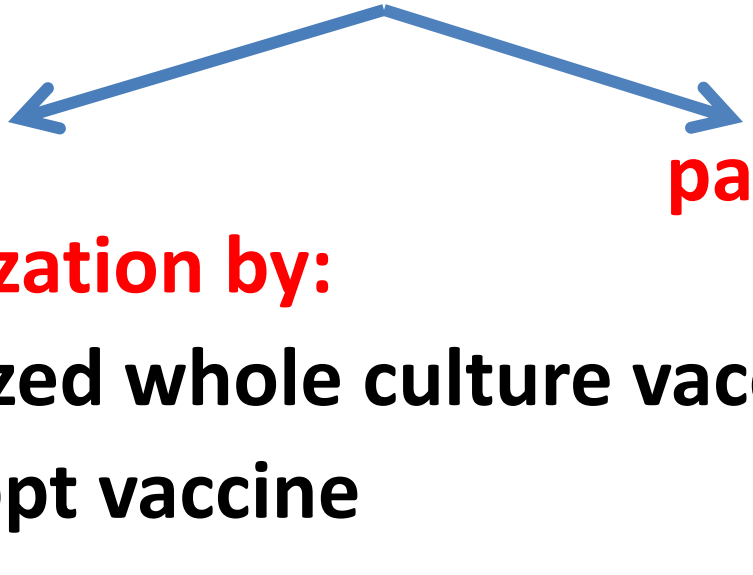
In case of fowl cholera must be differentiate from :

-New castle disease

-Fowl influenza

- Fowl typhoid or pullorum disease

Immunization & control



active

immunization by:

- **Formalized whole culture vaccine**
- **- alum ppt vaccine**

passive

of no value due to

rapid course of the disease

Haemophilus

- **Commensal organism of mucous membrane of animal & human**
- **Requires certain growth factors present in blood**
- **The most important species are :**

- H. influenza …….. Human

- H. agni …… avian

- H. parasuis …….. pigs

- H. paragallinarum ……. chicken

- H. somnus ……. bovine

morphology

- **Gram negative**
- **Small coccobacilli**
- **Non motile**
- **Non sporulated**
- **Non capsulated**

Cultural characters

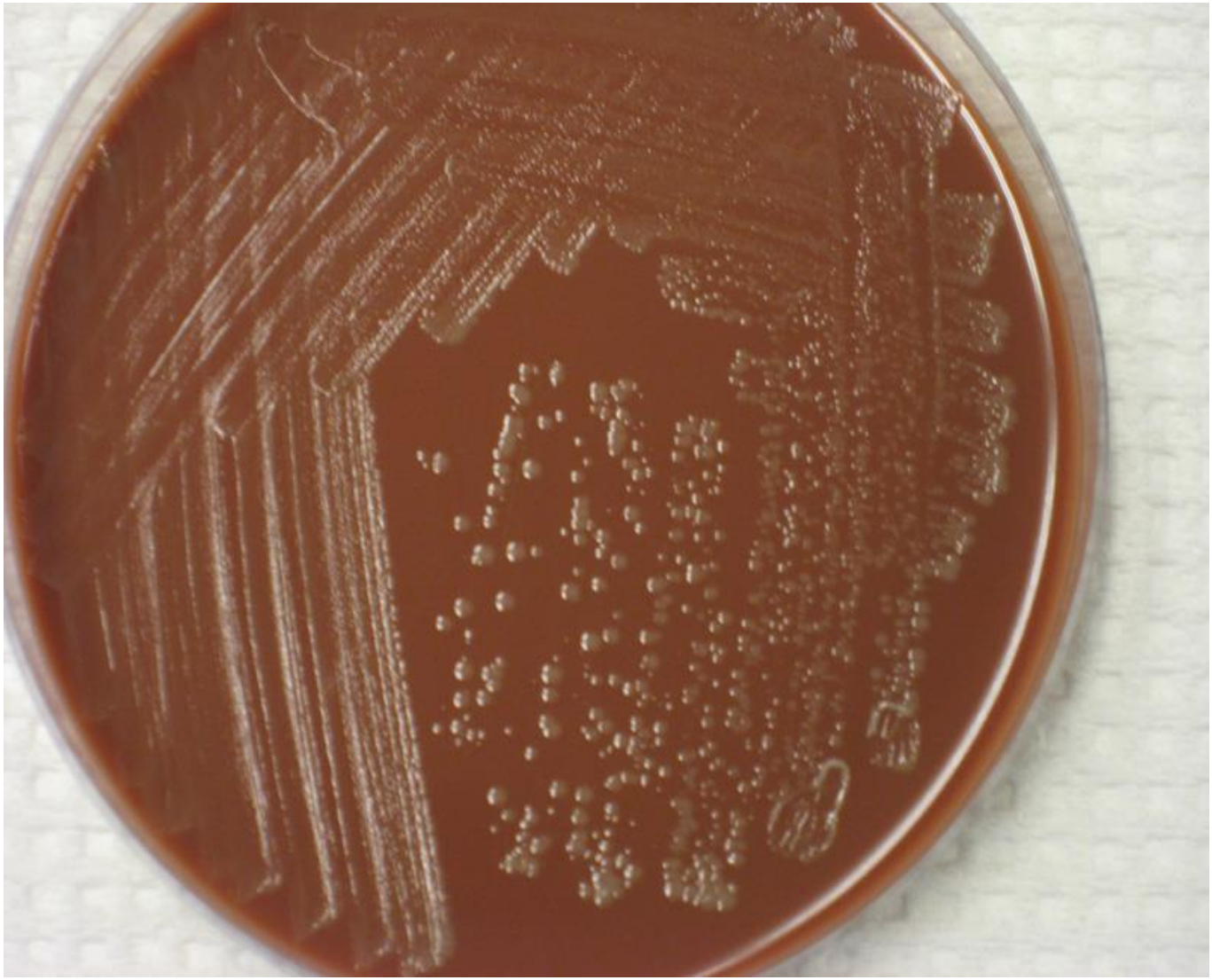
- **Facultative anaerobe at 37ºC**
- **Most strains require X & or V factor (in vitro)**
- **X-factor , heat stable(haemin) , V – factor , heat labile (NAD , Nicotinamide Adenine dinucleotide)**
- **Not grow on ordinary media**
- **Grow on blood agar & chocolate agar**

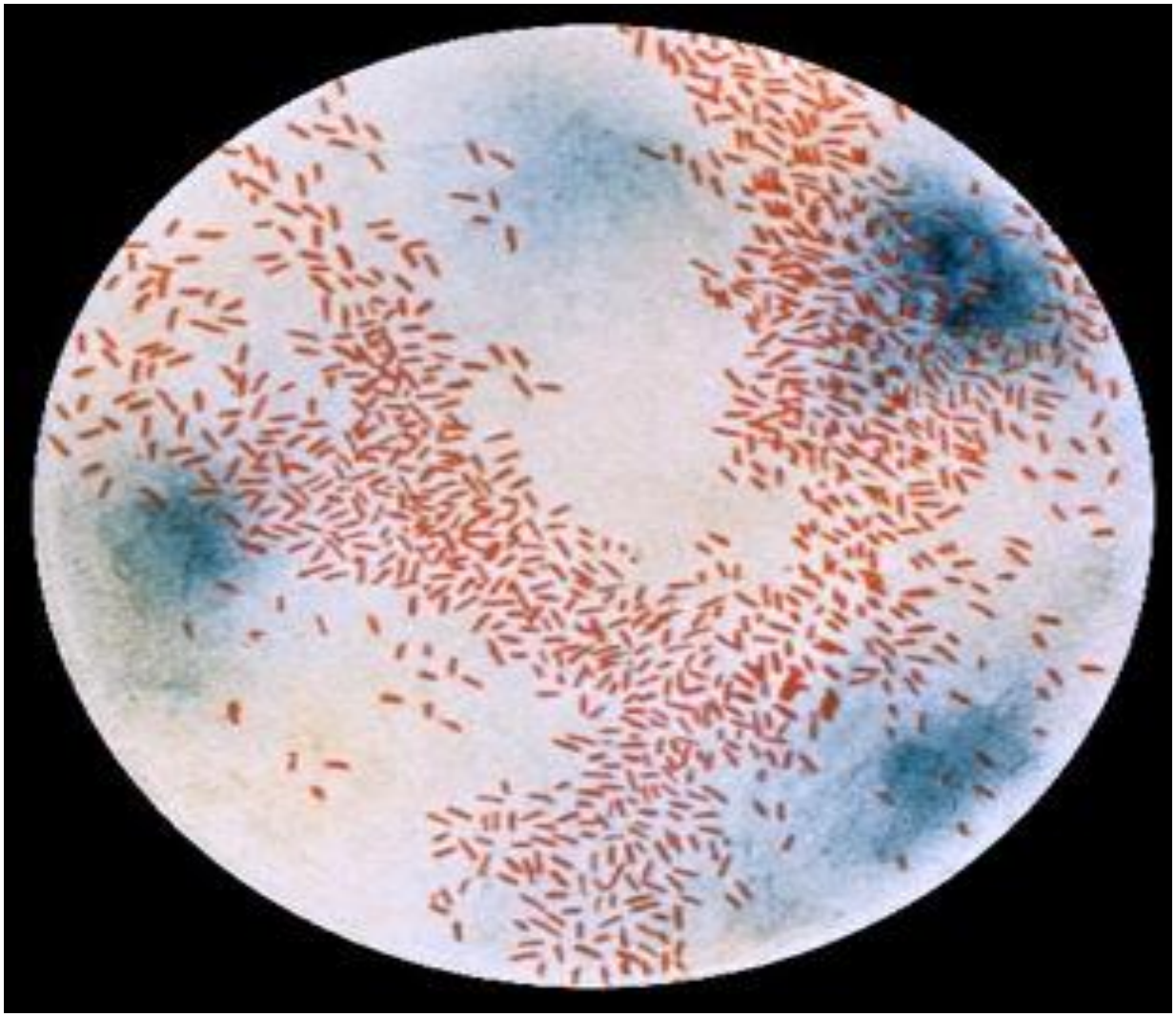
  vey small droplet like colonies

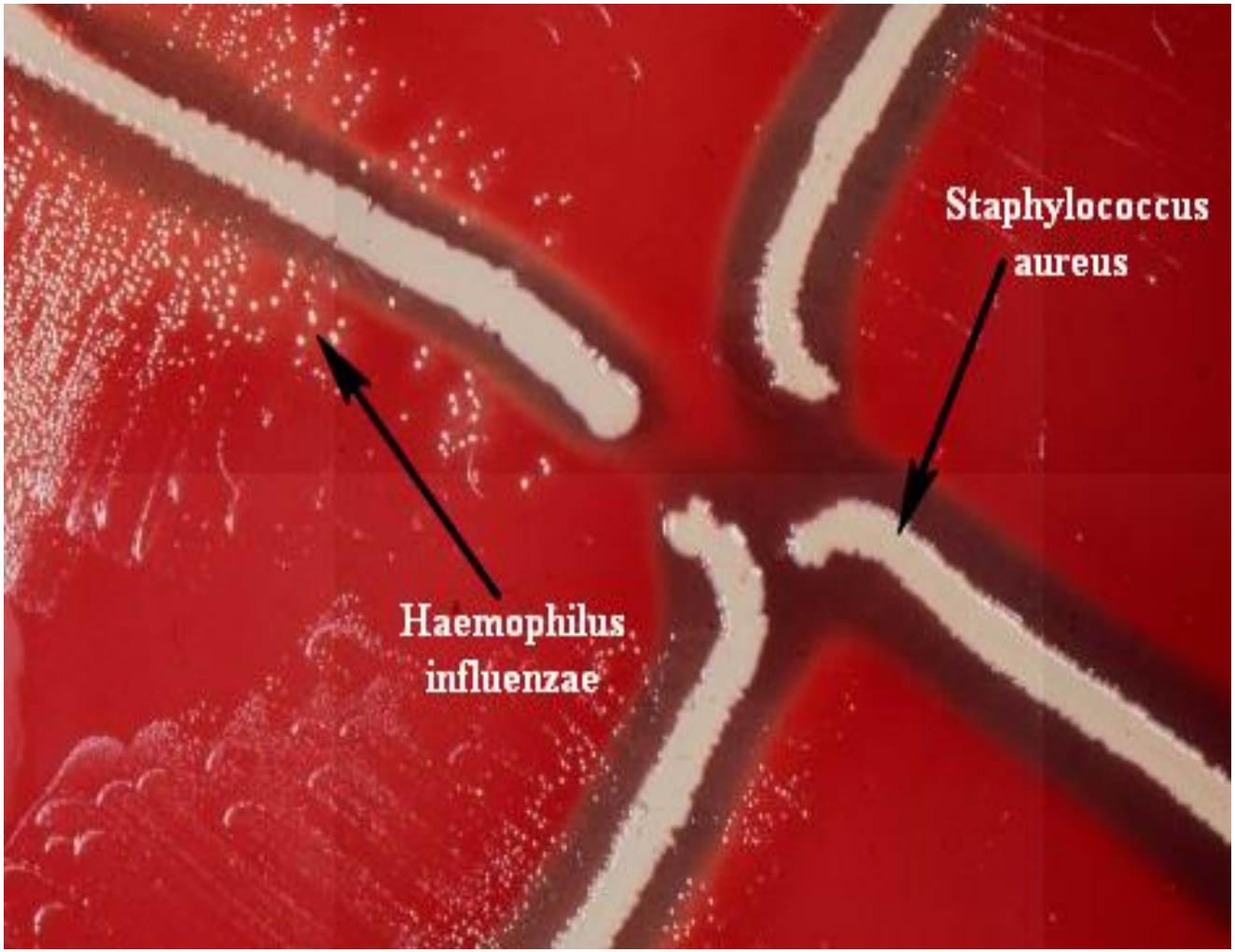
  some haemolytic & other non haemolytic

  smooth, translucent

With Stap.aureus streaking make satellitism phenomena






Staphylococcus
aureus
Haemophilus
influenzae

Biochemical characters

- Oxidase variable acc.to species
- Catalase -ve
- Sugar fermentation +ve (acid only)
- Citrate -ve
- Urea hydrolysis -ve
- H2S -ve

pathogenicity

H.paragallinarum → infectious coryza

H. influenza → otitis media,sinusitis, pneumonia & maningitis

H.agni → ovine septicemia,polyarthritis, pneumonia & meningitis

H.somnus → bovine thromboembolic meningeoencephalitis

Laboratory diagnosis

- 1- Isolation the organism on heated blood agar
- 2- Microscopic examination
- 3-Satellite phenomenon of blood agar treated with Staph.areus provide both X & V factor
- 4- Serological diagnosis by flourescent technique & counter immune electrophoresis

Order : Pseudomonales

- **Gram –ve rods**
- **Curved**
- **Spiral**
- **Occur singly**
- **Motile by polar flagella**
- **Differentiated to 2 families**

  family : Pseudomonadaceae

  family : Spirillaceae

Family : Pseudomonadaceae
genus : pseudomonas

pseudo = false

Monas = single unit

general characters:

- Widespread in nature
- In soil , water, plants & animals
- humans

3 members of pseudomonas of medical importance

- **P.mallei**
- **P.pseudomallei**
- **P.aeruginosa**

Pseudomonas mallei

- Actinobacillus mallei
- Malleomyces mallei

The cause of glanders الرعام

an infectious contagious disease of soliped animals & affect man

Characterized by nodules & ulcers on the skin, mucous membrane & internal organs

Burkholderia mallei
the causative agent of **glanders**, a dise
ase of horses
that is communicable to humans.
Previously called
Pseudomonas mallei.

Burkholderia pseudomallei
the causative agent of **melioidosis**,
a disease of rodents occasionally
transmitted to all domestic animal
species and humans. Formerly
called ***Pseudomonas pseudomallei***.

Morphology & staining reaction

- Gram –ve rods (long) pleomorphic
- Size : 0.5 – 1.5 – 4 µ
- Non sporulated
- Non capsulated
- Non motile
- **Easy to stain with carbol fuchsin (30 sec)**

granular appearance due to the storage of poly-β-hydroxybutyrate

Culture characters

- Grow aerobically pH 6.6
- Slowly on simple media
- Small colonies after 48 hrs
- Use of **enriched media** as :

  Heart infusion broth

  Trypticase soya agar

Or onto **selective media** : **1% glycerol broth**

uniform turbidity at 37 ºC (10 days) with the development of surface pellicle with brown colour + mucoid sedement

- **Onto glycerol potato agar**

  3 days incubation

  round, slimy, small pale yellow colonies

After 7 days **brown or choclate colour**

After 15 days **mucoid , sticky adhere to the media**

- **Biochemically inactive**

Clinical forms of glanders in horse

Pulmonary form	Nasal form	Cutaneous form (farcy)
1- coughing 2- bloody stained mucus 3- rise of temperature	1-Redness of nasal Mucosa 2-Mucoid ,purulent bloody discharge 3-Ulcer on the nasal mucosa & septum nasi	1-S/C nodules along the lymph vessels (farcy buds) 2-Ulcer with yellow oily pus 3-The local lymphatic vessels become corded

It is called glanders:
when the principal lesions are seen in nostrils and sub-maxillary glands and lungs

It is called farcy :
when lesions are on the surface of limbs and body

Laboratory diagnosis

- **History & sampling**

Symptoms

Closed nodules under complete aspetic condition

- **Bacteriological examination**

  if necessary onto glycerol potato agar

- **Serological test :**

- Agglutination test 1/1000 +ve
- Complement fixation test (human)

- **Animal inoculation**

Strauss reaction in G.pig:

By inoculation of the discharge in O G.pig I/P

+ve strauss reaction → orchitis after 10 -14 days

- **Differential diagnosis : in horses**

- **Strangles → Streptococcal infection**

- **Ulcerative lymphangitis → C. ovis**

- **Epizootic lymphangitis → histoplasma**

Allergic test

- Based on the delayed hypersensitivity
  similar to tuberculin test

Named mallein test:

mallein : extract of B.mallei which grown on glycerol broth 8 – 10 weeks

then killed by boiling 2hrs

evaporate to 1/10 (conc mallein)

Act as diagnostic antigen

Application: by intradermal palpebral test

0.1 ml of mallein in the skin of the lower eye lid near the mid canthus of the suspected animal

After 24 -48 hrs

+ve result **oedematous swelling of the eye lid**

closed eye

severe lacrimation

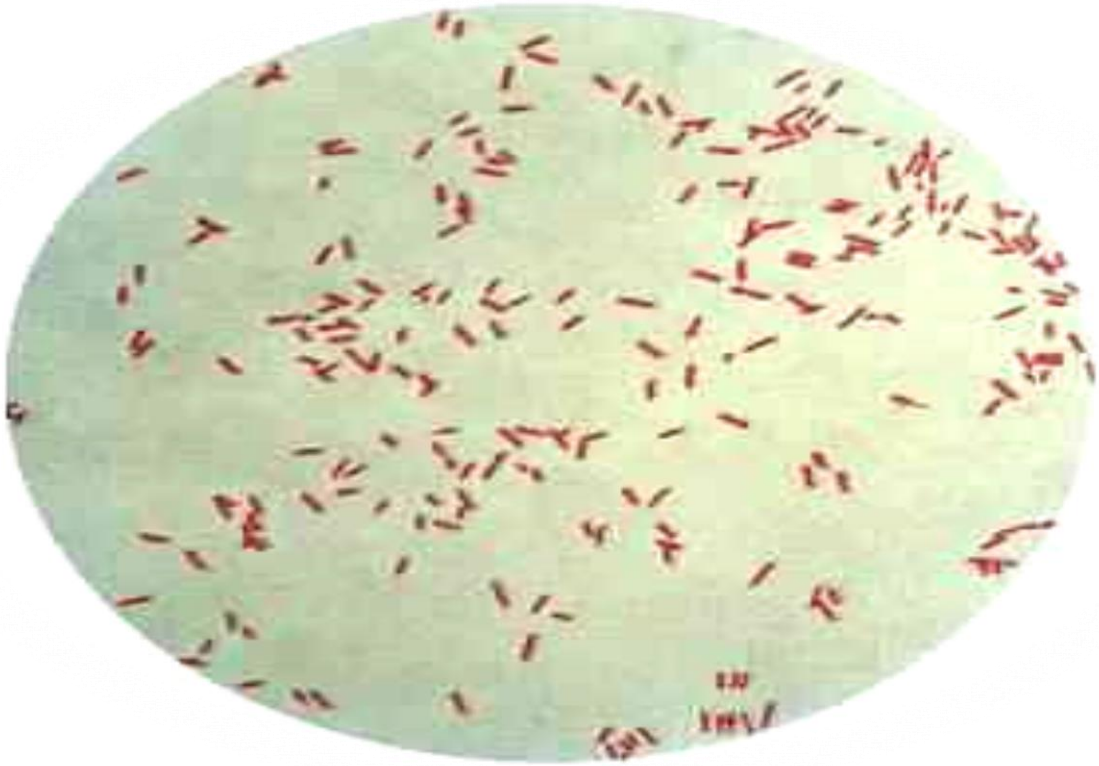
purulent discharge

Pseudomonas aeruginosa

- Wide distribution
- Associated with a wide variety of suppurative infections in man & animals
- It is a part of the normal intestinal flora
- Characterized by green pus

morphology

- Rod shape
- Size 1.5x 0.5 µ
- Actively motile by polar flagella
- Non capsulated
- Non sporulated
- Gram –ve , sometimes not easily decolorized



Cultural characters

- Strict aerobic
- pH neutral
- Grow on ordinary media at 37ºC
- Growth range 5-43ºC

- **Nutrient broth** **nutrient agar**

After 18 hrs convex, irregular
Turbidity+bluish green edges, greyish
Colour + sickly odour + colonies &
Pellicle + thick mucoid sed. pigmented

Ps. aeruginosa
Nutrient agar

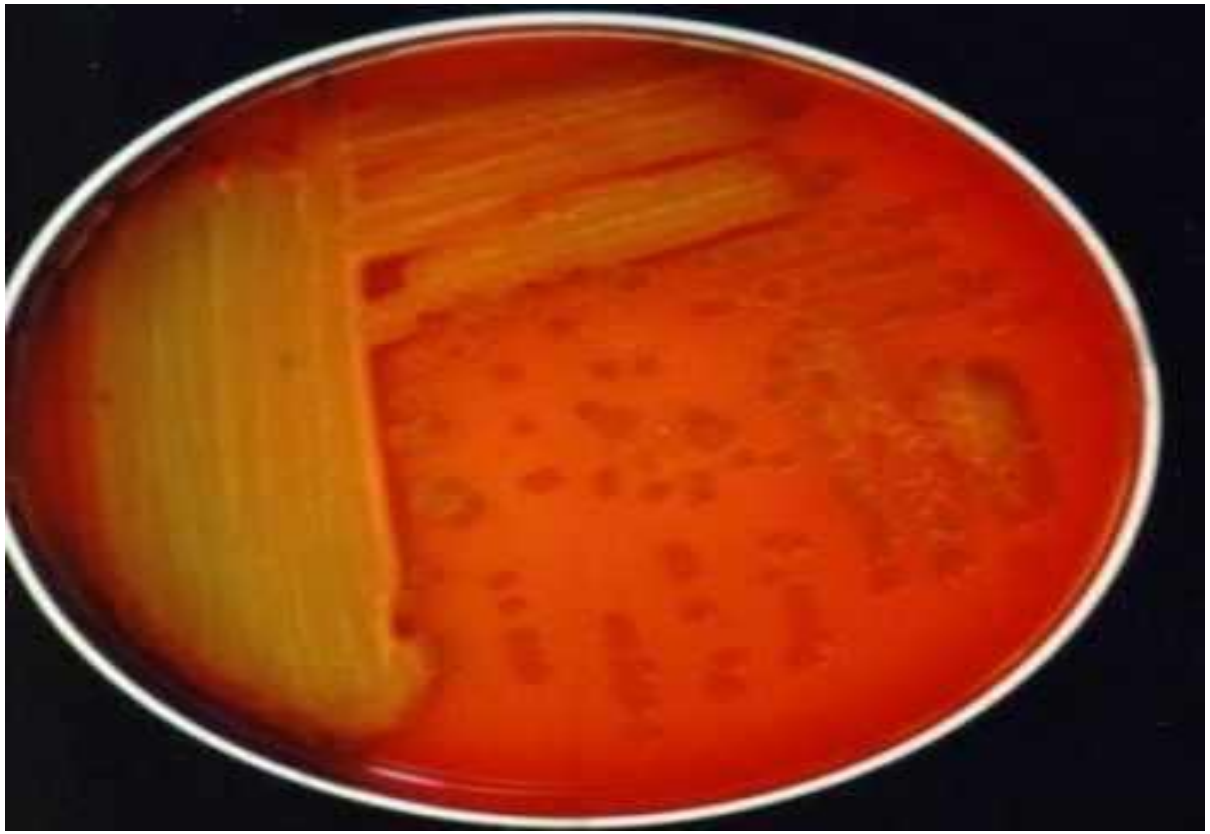
- P. aeruginosa produce 2 types of water soluble exogenous pigments

- Pyocyanin blue pigment
- Fluorescin green pigment

- **Onto blood agar** : large flat colonies
  haemolytic (complete zone)

1- **S-form** (soft , shiny & smooth
2- **R-form** (dry granular)
3- **M-form** (mucoid confluent)

Onto MacConkey agar : pale , non lactose fermenting bacteria



Biochemical reactions

- Oxidase +ve
- Catalase +ve
- Glucose oxidation +ve
- Glucose fermentation –ve
- Lactose - ve
- Indole -ve
- MR -ve
- VP -ve
- **Citrate +ve**
- **Urease +ve**
- **H_2S -ve**
- Nitrate reduction -ve
- **Gelatin liqification +ve**

Antigenic structure

O – antigen & H – antigen are recognized by :

agglutination test

into 29 serotypes

- **Ps.aeruginosa (pyocynogenic strains) produce :**

1- pigments → **pyocyanin** (bluish-green ,has antibiotic effect on different organisms, haemolytic activity)

→ **fluorescin** (pyoverdin) (yellowish green ,slight soluble in H2O& insol. In chloroform)

→ **pyorubin** (red pigment, rare)

2- Proteolytic enzymes

3- Bactericidal substances

4- Pyocyanase :

thermostable exotoxin,

haemolytic activity

bactericidal activity

antigenic & lipoid in nature

Epidemiology & pathogenesis

- Ps. Widely distributed in nature, soil & water
- Ps. Is a facultative pathogen

- **Bovine** : generalized disease, mastitis, respiratory diseases, enteritis, synovitis, arthritis, lung & liver abscesses

Horse : infections of reproductive organs, sterility, abortion, foal mortality, lung abscesses

Pig : enteritis, respiratory infections, otitis

Dog& cat : generalized diseases,pneumonia, otitis externa, endocarditis,urethroadenocystitis infections of the reproductive organs

Laboratory animals : generalized infections , pneumonia , enteritis

- **Reaction type I a**

Oxidase – positive fermentative bacteria

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ملف مناعة رقم إثنان.pdf

Mycoplasma lecture.pdf

د. محمد وهيبه.pdf

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16 photos • 11 June 2021

Adaptive immunity

1

12:37 pm

- specific defence mechanism,
- Effective against a specific M.O -
- depend upon having previous contact - with a particular M.O (MEMORY)

2

12:37 pm

Acquired immunity

Active			passive	
Natural	artificial		natural	artificial
-clinical & subclinical infection			transplacental or colostrum	
vaccination for prophylaxis			administration of immune serum or cells	
built after long period			immediately	
Of long duration			short	
- used for prophylaxis -Due to antigen administration			for treatment & prophylaxis Ab adminis	

16

Humoral immunity is more important than cellular immunity in defending against microbes with capsules rich in polysaccharides & lipids, & against polysaccharides & lipid toxins .

The reason for this is :

B cells respond to& produce antibodies specific for many types of molecules but T cells , the mediators of cellular immunity recognize & respond only to protein antigens

15

Fundemental distinctions in vivo

	Humoral immunity	Cellular immunity
rate of response	faster	delayed
Method of transfer	Transferred passively by serum	Transferred only with special cells

14

CD4+ Helper T Cells :

Activate phagocytes to destroy microbes residing in the vesicles of these phagocytes

CD4+ Cells + microbes → cytokine secretion → inflammation → macrophage activation , killing of ingested microbes

CD8+ T lymphocytes cells :

Kill any cell containing microbs or microbial proteins in the cytoplasm , thereby eliminating the reservoir of infection

13

Cell mediated immunity (Cellular immunity)

It participates mainly in infections by agents that - enter the cells as T.B , brucella , viruses & fungi -

T – lymphocytes are the cells of cell mediated immunity, ◦

Cell mediated immunity is the arm of the adaptive Immunity ◦

That combats intracellular microbes which are ingested by ◦ phagocytes & live within these cells or microbes that infect non-phagocytic cells

9

antibodies induce resistance through :

1) opsonize the M.O i.e combine with surface antigens that interfere with phagocytosis (Ig G)

2) Agglutination the M.O , so become easily phagocyted (Ig M)

3) Direct bactericidal or lytic effect with complement (Ig G, IgM)

7

secrete - it depend on T- lymphocytes when sensitized , it - several soluble products known as lympehokines (following functions :interleukines) that carry the

1) cytotoxic destruction of the invading cells by T- cytotoxic - cells

2) mononuclear inflammatory reaction (chemotaxis & - activation of macrophages

3) cytotoxic destruction of Ab coated cells by killer - cells ADCC

10

The responses of T-lymphocytes consist of squential - phases :

1- recognition of cell associated microbes by naive - T-cells -

2- expansion of the antigen specific clones by - proliferation

3- differentiation of some of the progeny into effector - cells & memory cells

11

The responses of T-lymphocytes consist of squential - phases :

1- recognition of cell associated microbes by naive - T-cells -

2- expansion of the antigen specific clones by - proliferation

3- differentiation of some of the progeny into effector - cells & memory cells

11

4) Block the infective ability of the M.O or virus (Ig G & Ig A)

5) Neutralize toxin or cellular product

6) Precipitate toxin or cellular product

8

Most of antigens must be first processed & - presented by the antigen presenting cells (APC) to the CD4 cells

- APC produce interleukin 1 which stimulate CD4 - TH Cells to produce interleukins 2 & 4 & 6

Humoral immunity

- through the production of antibodies against the M.O or their products

Antibody generation depend on activation of - B-lymphocytes → plasma cells → Ab - required

- a few numbers of antigens can directly activate B- - cells such as antigens are called thymus independent antigens or mitogens

Adaptive immunity

1

- specific defence mechanism,

- Effective against a specific M.O -

- depend upon having previous contact - with a particular M.O (MEMORY)

2

M.O that overcome the innate non specific immunity come up against host' s second line of defense

I.e. antigens of the invading M.O come in contact with cells of the immune system

(macrophages & lymphocytes)

acquired specific immunity make up by 2 mechanisms

Humoral → production of antibodies

cellular → production of lymphokines & cytotoxic effect

3

Humoral immune response

- Humoral immunity is mediated by antibodies
- The arm of the adaptive immune response
- That neutralize & eliminate extracellular microbes & microbial toxins

4

immunity

Immunity provides

protection against organisms,

the functions of the immune system are .

recognition of these foreign pathogens and

defence of the body against them

Defence is mediated by a range of innate & adaptive cells & molecules that stimulate inflammation& cause digestion or lysis of foreign pathogens or infected cells

Antigens (immunogens(

They are substances capable of stimulating a specific immune response

simple or compound nitrogenous -substance

:

_in order to be antigenic,

the substance must fullfil certain conditions

-1foreignness:

Immune system recognize (self antigens) does not get

activated against them

_more foreignness the substance has the antigenic it will be re powerful

-2molecular size

Substance with molecularweight over 100,000 daltons are antigenic

Around 10,000 are weak ones

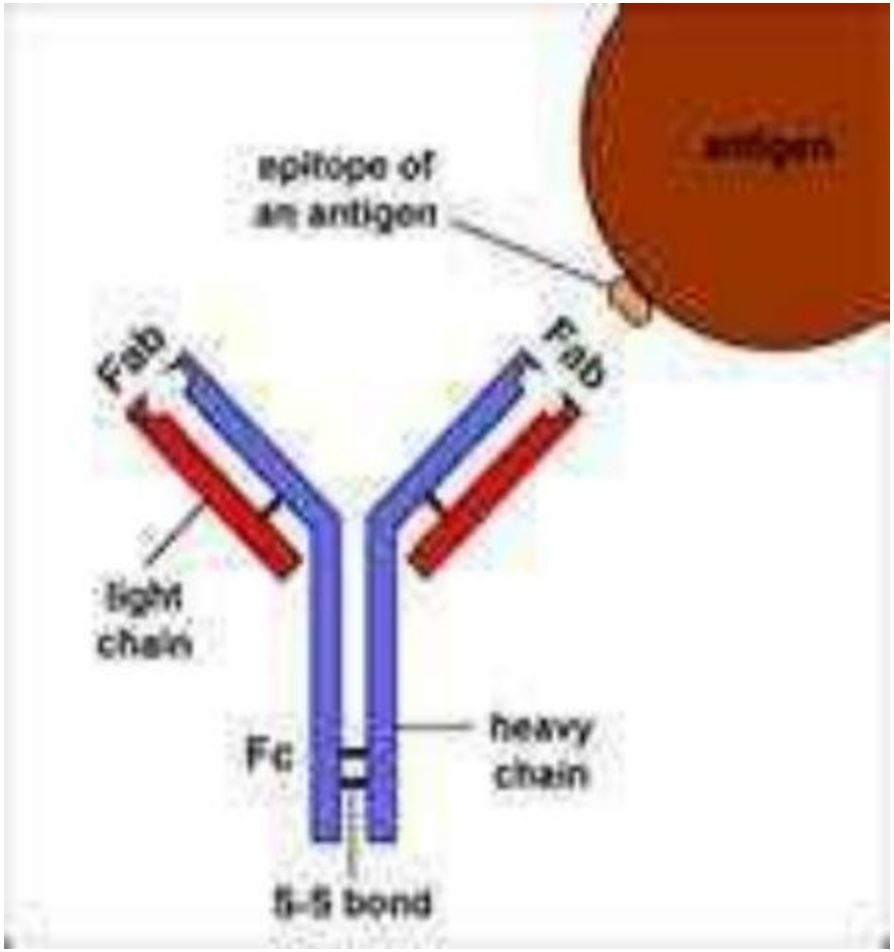
Monosaccharides & amino acids are not immunogenic,

while protein & polysaccharides?????? are immunogenic

-3specificity

Strong antigens are large molecules- , however only a restricted portions of them , are involved in the actual binding with the antibodies

-antigenic determinants (epitopes) - determine the specifity of the antigen


antigen
epitope of
an antigen
Fab
Fab
light
chain
Fc
heavy
chain
S-S bond

-4chemical complexity

Molecules must posses certain degree of chemical complexity in order to be antigenic-

-5others

Route of adminstration-

-presence of adjuvantstimulate immune response better -

Antigenic types

-ıheterologous antigen:

Present in different animals & different tissues

Ag derived from one species & can stimulate response in another species

e.g insulin that was produced from pig used by human to treat diabetes

-2homologous antigen:

Where specific antigen react with specific antibody

-3auto antigen:

It is a self antigen (normal constituent of the body tissue) no immune response is formed against them

-4iso antigen:

Present in R.b.Cs & responsible for blood grouping

-5forsman antigen:

Presentin G.pigs tissues & R.B.Cs

Also in human , rats, dogs but absent in cows, rabbits & pigs

Have the ability to react with different antibodies in the same animal tissues

Antigenic structure

	immunogens	hapten
Chemical structure	Protein A.A Mucopeptides	fats CHO others
	Able to stimulate an immune response& production of antibodies	Able to react with antibodies Unable to stimulate their production directly
Molecular weight	high	Less than 5000 daltons
	Responsible for stimulation	Responsible for specification of antibodies

Epitope:

-Small part of an antigen that interacts with an antibody. 10-12 amino acids

-Any given antigen may have several epitopes.

-Each epitope is recognized by a different antibody.

Bacterial antigens

There 5 bacterial antigens:

-1somatic Ag (O: (

In cell wall & cytoplasm

Protein in nature

Heat resist

Formaline sensitive

Alcohol or phenol resist

Flagellar antigen (H(

Flagellin (protein in nature(

Heat sensitive at 6o -8o °c

Alchol & phenol sensitive

Formaline resist

-3capsular antigen (K(

Polysaccharides, mucopeptides, polypeptides

Heat resist

Present in capsulated bacteria only

Surface antigen (S:(

Polysaccharides

Present in streptococci , E.coli, pasteurella

-4virulence antigen (Vi(

Characteristic for) Freshly cultivated) diphetria bacilli

Simillar to surface antigen

Repeated cultivation ⟶ the virulence disappear (salmonella bacilli(

-5spore antigen

In sporulated bacteria only

Immunoglobulins (antibodies(

General characters:

-1glycoprotein

-2present in serum & body fluids

-3induced when immunogenic molecules are introduced into the host'slymphoid system

4- reactive with, and bind specifically to, the antigen that induced their formation

-5All immunoglobulin molecules have a common structure of 4 polypeptide chains
2 large or heavy chains.

2small or light chains.

-6There are 5 classes of human immunoglobulins.

-7The difference between them depending on their heavy chains and called isotypes.

-8Ig heavy chains are designated

IgG gamma Y chains
IgM mu μ chains
IgA alpha α chains
IgE Epsilon Є chains
IgD delta δ chains

-9Light chains

Kappa κ

Lambda λ

How Do B Cells Produce Antibodies

?

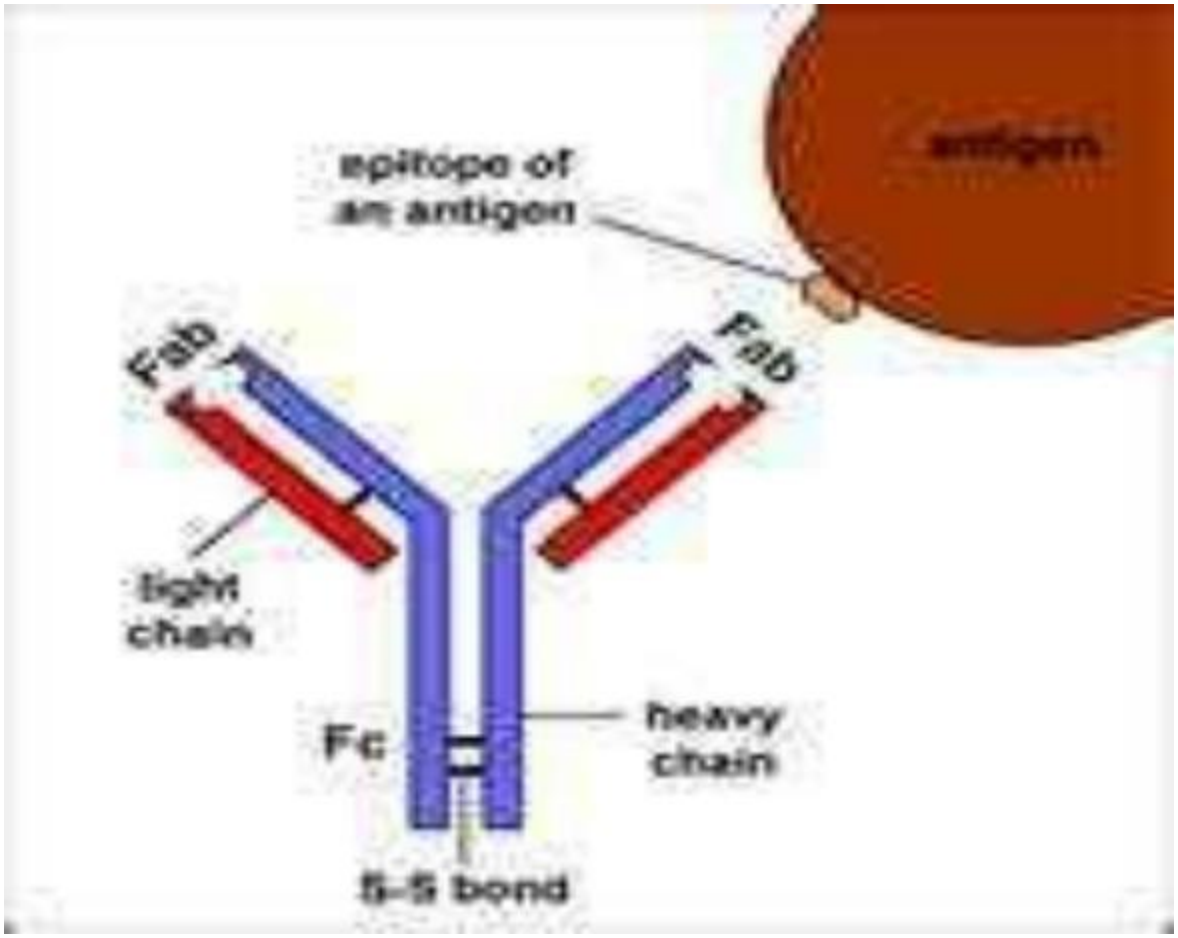
B cells develop from **stem cells** in the bone marrow of adults (liver of fetuses.(

After maturation B cells migrate to lymphoid organs (lymph node or spleen.(

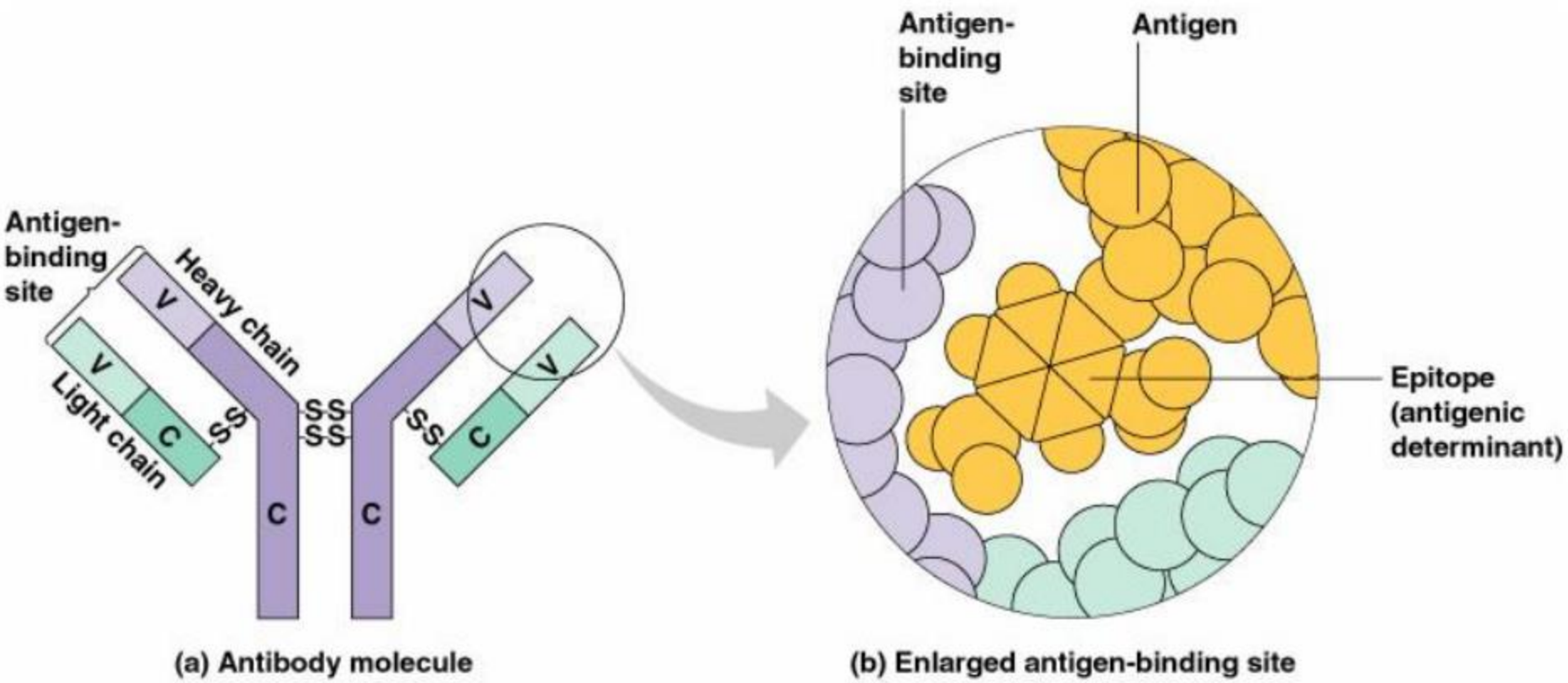
Clonal Selection: When a B cell encounters an antigen it recognizes, it is stimulated and divides into many clones called **plasma cells**, which actively secrete antibodies.

Each B cell produces antibodies that will recognize only one antigenic determinant.

Antibody structure



Antibody Structure



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Heavy Chain – 110 amino acids long

100 distinct V segments

30 D segments

6 J segments

Enzymes choose one V segment, one D segment and one J segment and fuse them together

18,000 combinations in encoding antibody molecule

this variable region to the constant region

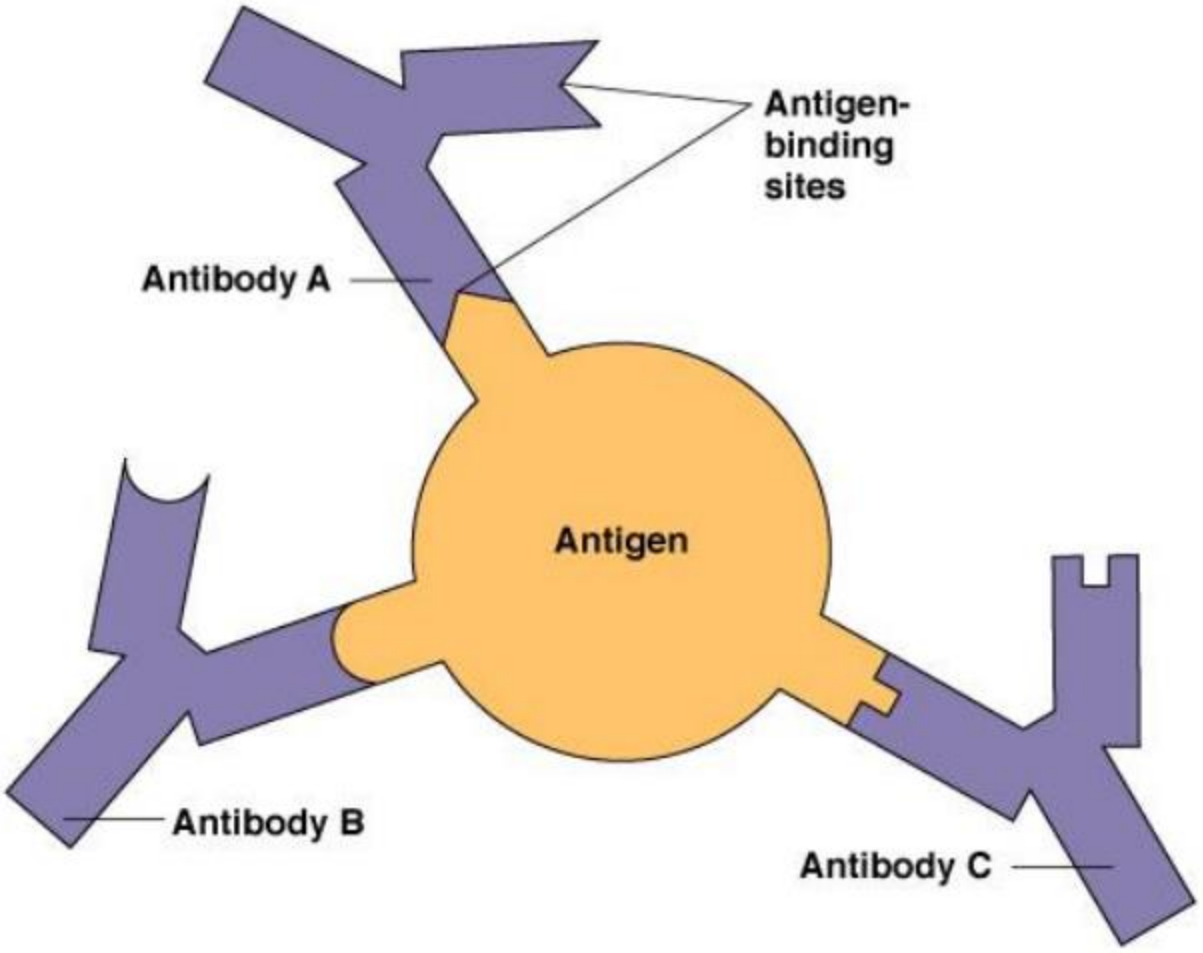
Light Chain — 211 amino acids long

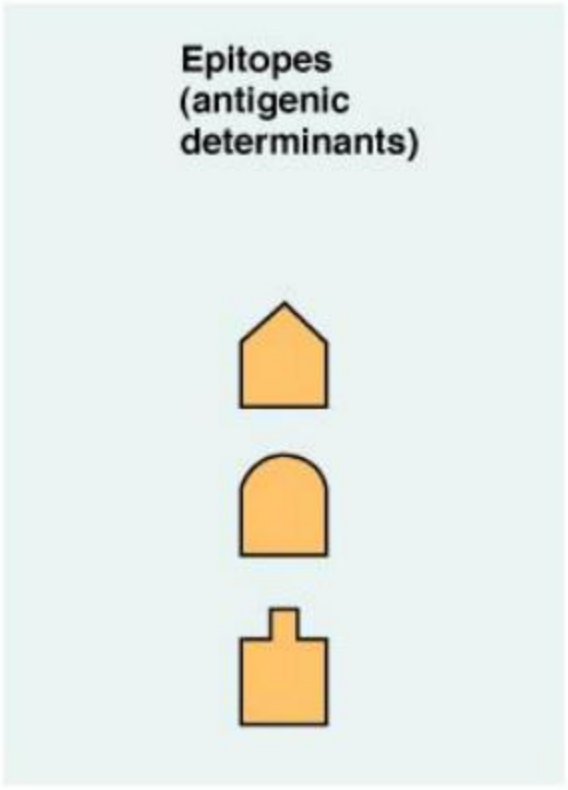
10,000 combinations

Total of 180,000,000 distinct B cells

Fusion is sloppy, can create other variants

Epitopes: Antigen Regions that Interact with





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Ig G

The major immunoglobulin of serum - 75%

-molecularweight is 150000 in humans-

-the secondary response antibody-

4 -subclasses are found IgG1 , IgG2, IgG3, IgG4 -

Differed in- :

their concentrations- ,

aminoacid composition- ,

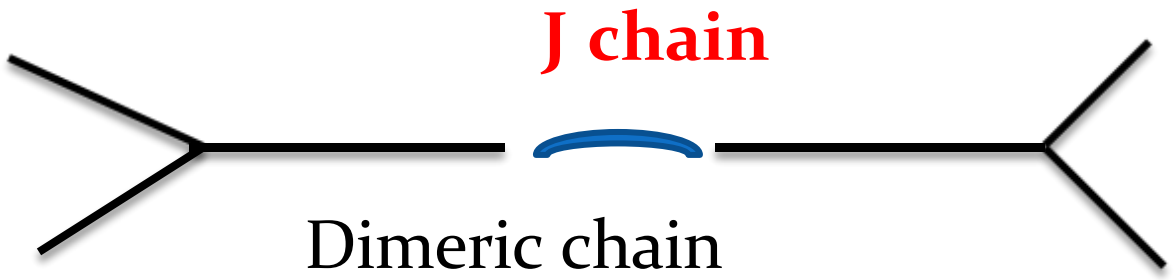
Number & position of disulphide bonds-

Biological functions-

IgA

Most of the serum IgA occurs as a monomer, but in many other mammals found as dimer

The dimer is held together by J chain



Produced by the antibody producing plasma cells-

-IgA is the predominant antibody class in seromucous - secretions such as saliva , tears, clostroum, respiratory, gastrointestinal & genitourinary secretions

-it facilitates passage through the epithelial cells- & protects the secreted molecule from proteolytic digestion

-there are 2 subclasses of IgA (IgA 1 , IgA- (2

Ig M

- Is a pentamer of the basic unit with a μ heavy chain & a single J chain

- -is the first antibody type to be produced during an immune response

Ig D

Many circulating B cells have Ig D present on their surface

-less than 1% of the circulating antibody-

-have a short half- life in serum because the protein is very susceptible to proteolytic attack

Ig E

-Is present in extremely low levels in the serum

-It found on the surface of the mast cells & basophils

The immune system is split into 2 functional divisions:

Innate immunity

natural or native

the first line of defence against infectious agents

acquired or adaptive

specific response to each infectious agent

Innate immunity

All multicellular organismscontain intrinsic mechanisms of defence against infections, which constitute innate immunity

The mechanism of innate immunity respond to microbes & not to non microbial substances

Are specific for structures present on various classes of microbes

- **are mediated by receptors encoded in the germ line**
- **are not enhanced by repeat exposure to microbes**

The principle components of innate immunity

-1epithelia:

provide physical barriers against microbes, produce antibiotics & contain lymphocytes that may prevent infections

-2the principle phagocytes, neutrophils & monocytes - macrophages are blood cells that are recruited to sites of infection

Neutrophils & macrophages recognize microbes by several receptors, ingest the microbes, secrete cytokines)

elimination of microbes & repair of infected tissues(

-3natural killer cells NK cells:

Kill host cells infected by intracellular microbes & produce the cytokine which activates macrophages to kill phagocytosed microbes

-4the complement system:

Is a family of proteins that areactivated sequentialluyon encounter with some microbes & by antibodies (in the humoralarm of adaptive immunity(

Complement proteins coat (opsonize) microbes for phagocytosis, stimulate inflammation, & lyse microbes

-5cytokines of innate immunity function to:

-Stimulate inflammation

-Activate NK cells

-Activate macrophages

-Prevent viral infections

Adaptive immunity

Cell mediated immunity

T - lymphocytes are the cells of cell mediated immunity,

Cell mediated immunity is the arm of the adaptive Immunity.

That combats intracellular microbes which are ingested by phagocytes & live within these cells or microbes that infect non-phagocytic cells

The responsesofT-lymphocytes consist of - squential phases:

-1recognition of cell associated microbes by - naive

T-cells-

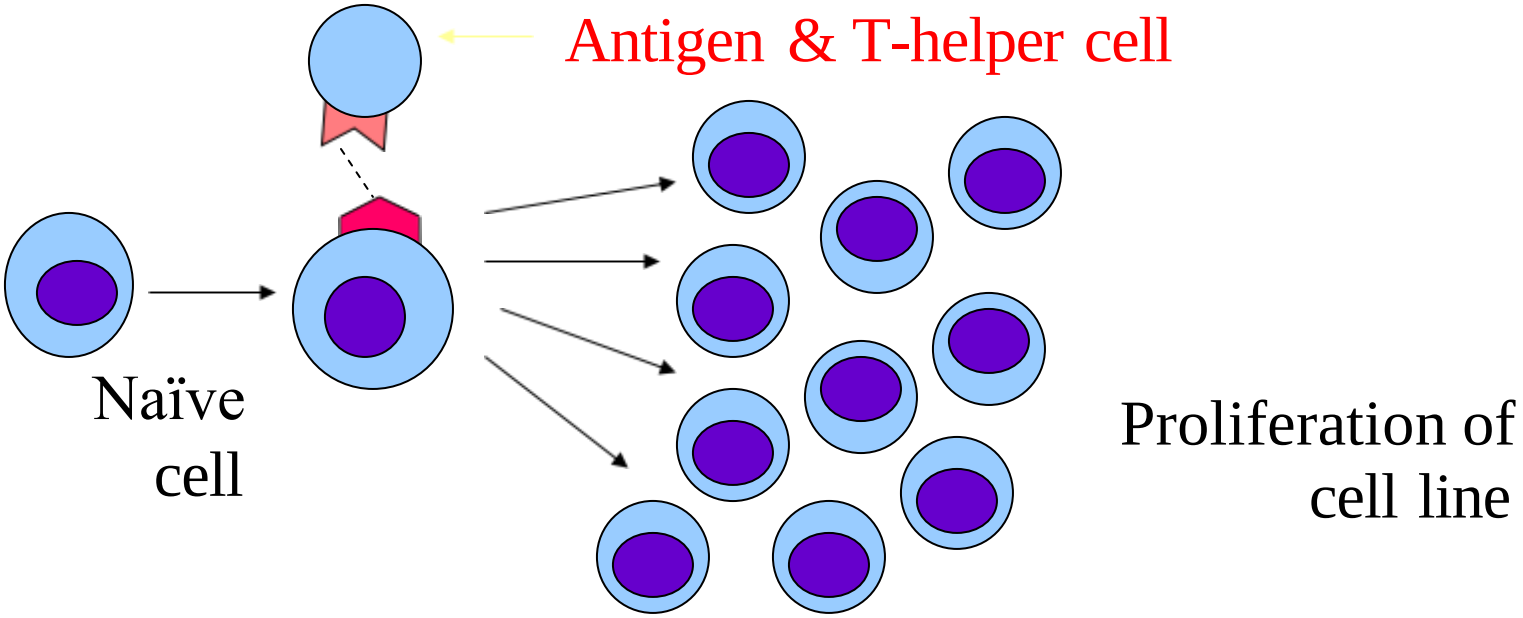
-2expansion of the antigen specific clones by - proliferation

-3differentiation of some of the progeny into - effector cells & memory cells

When help arrives. . .

The T-helper cell receptor "docks" with the B cell's MHComplex

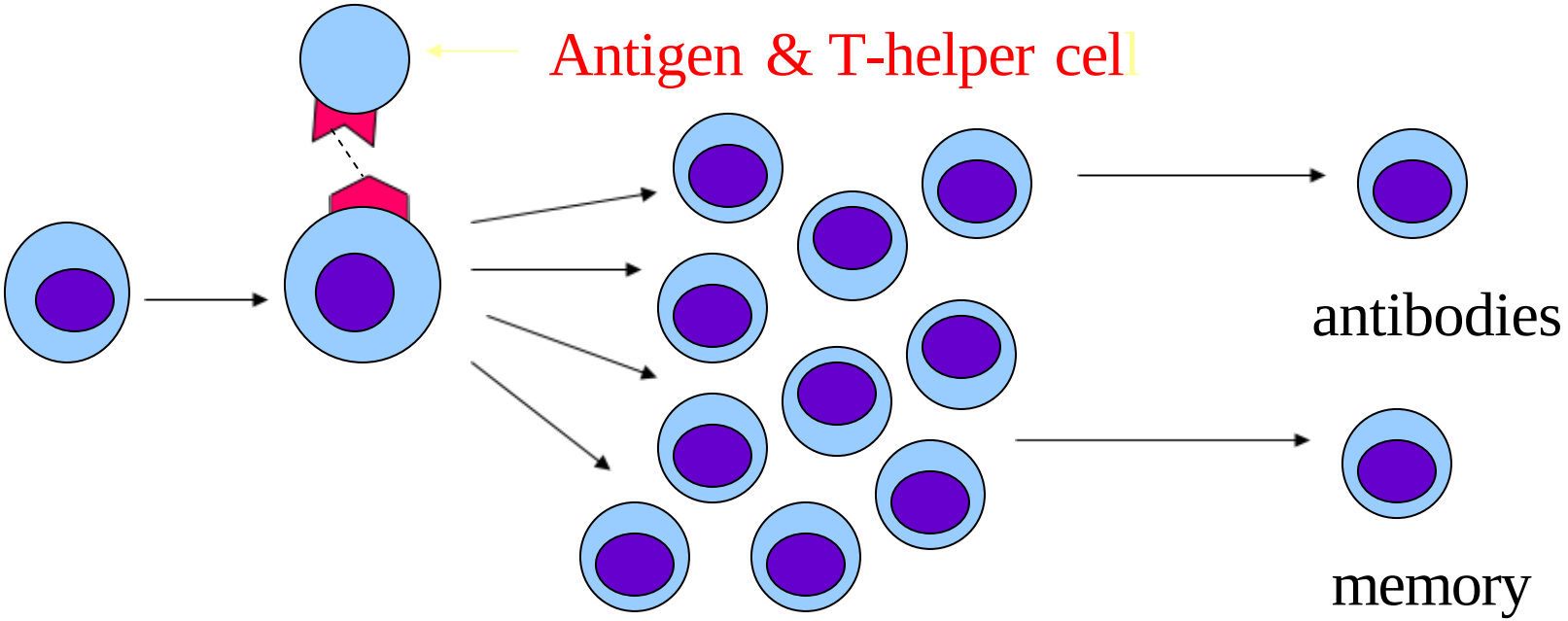
B cells proliferate. . . .



B cells differentiate into. . .

Antibody producing cells [attack mode. [

Memory cells [remembers & future protection. [



Types of cell mediated immunity

There are 2 types of cell mediated immune reactions:

CD4+ helper T cells

CD8+ cytotoxic T lymphocytes

CTLs

CD_4+ Helper T Cells:

Activate phagocytes to destroy microbes residing in the vesicles of these phagocytes

CD_4+ Cells + microbes → cytokine secretion

inflammation **macrophage activation , killing of ingested microbes**

CD8+ T lymphocytes cells:

Kill any cell containing microbs or microbial proteins in the cytoplasm , thereby eliminating the reservoir of infection

Humoral immune response

Humoral immunity is mediated by antibodies.

The arm of the adaptive immune response.

That neutralize & eliminate extracellular .
microbes & microbial toxins

Humoral immunity is more important than immunity in defending against microbes cellular with capsules rich in polysaccharides & lipids, & against polysaccharides & lipid toxins.

The reason for this is:

B cells respond to& produce antibodies specific for many types of molecules butT cells , the mediators of cellular immunity recognize & respond only to protein antigens

Hypersensitivity

Def:

Excessive or inappropriate response to an antigenic stimulus (bacteria, protein, hapten, serum or some drugs) previously the body infected with them.

harmful to the host

This reaction is rapid or slow after allergy

There are 4 types of hypersensitivity

-1anaphylactic:

If a G.pig is injected with a small dose of an antigen (egg albumin) → no adverse effects are noted
If a second injection of the same antigen is given intravenously after an interval of about 2 weeks

Symptoms : asthma leads to death

Ig E produced in this case

A condition known as anaphylactic

Mechanism

Antigen stimulates formation of AB (IgE- (

-IgE fixes, by its Fc portion to mast cells and-

basophiles (cell bound IgE- (

Re-exposure to the same sensitizing antigen (allergen), it - cross – links the Fab portion of mast cells and release of preformed mediators (Immediate phase): within minutes, vasodilatation ……., ready preformed mediators)histamine(

Cytokines produced by mast cells (TNF, IL-4& IL- (5 stimulate the recruitment of leukocytes, which cause the late phase reaction (6hours after exposure, erythema, -edema……..broncospasm . Mediators (leukotriens.(

-2type II : cytotoxic

Type II reactions are initiated by the binding of an antibody (IgM & IgG)to an antigenic component on a cell surface (drug , microbial product passively absorbed onto a cell surface(

The cell that is covered with antibody is then destroyed by the immune system

Ex.: salmonella & mycobacteria are associated with haemolytic anaemia

Due to an immune reaction against alipopolysaccharide bacterial endotoxin that coated onto erythrocytes of the patient → anaemia

Mechanism

Activation of complement.

Phagocytosis : mediated by opsoninson cells which.
detected by receptors on phagocytes

ADCC by Nk.

-3type III : immune complex

Sometimes , antibodies (IgM & IgG) against soluble antigens may form complexes with the antigens & the immune complexes may deposit in bloodvessels in various tissues , causing inflammation&
tissue injury

Such diseases are called immune complex diseases & represent type III hypersensitivity

For example : Pencillin & sulphonamides for susceptible patients

Type IV cell mediated or delayed

Cell mediated hypersensitivity are seen in a number of- chronic diseases due to mycobacteria, protozoa& fungi-

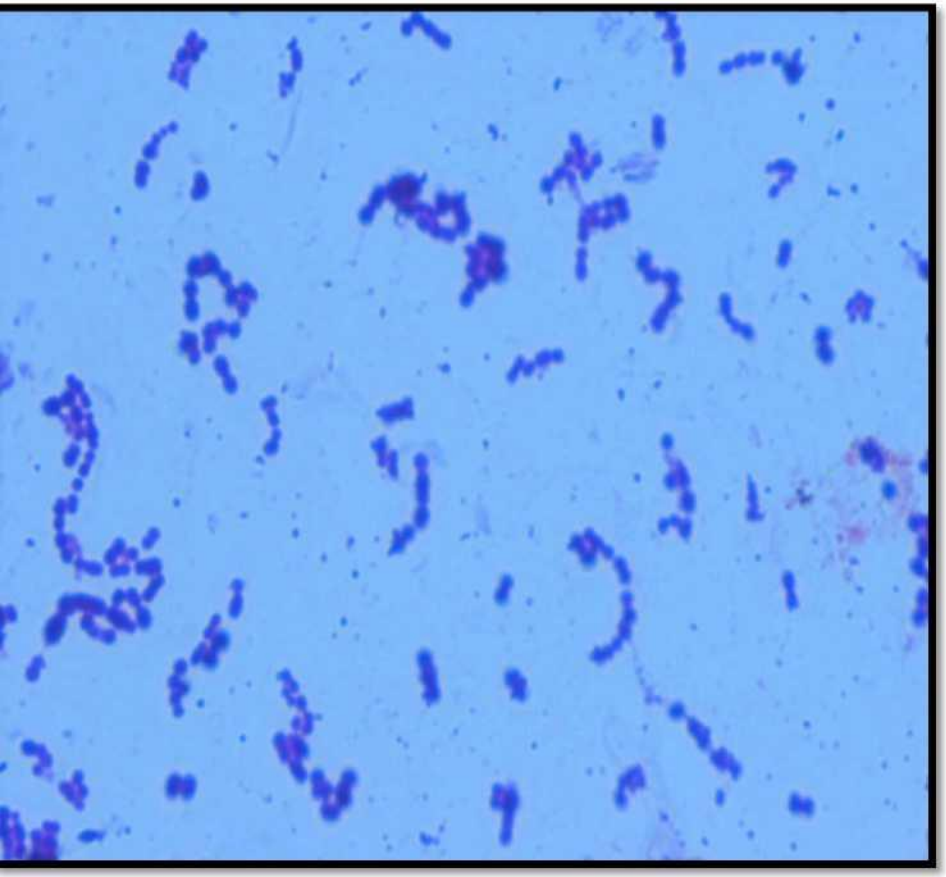
Because the host is unable toelimenate the micro-organism the antigens persist & give rise to a chronic antigenic stimulus.

Continual release of lymphokines from sensitized T - lymphocyte cells (Th & Tc)results in the accumulation of large numbers of activated macrophages

These cells can fuse together to form giant cells

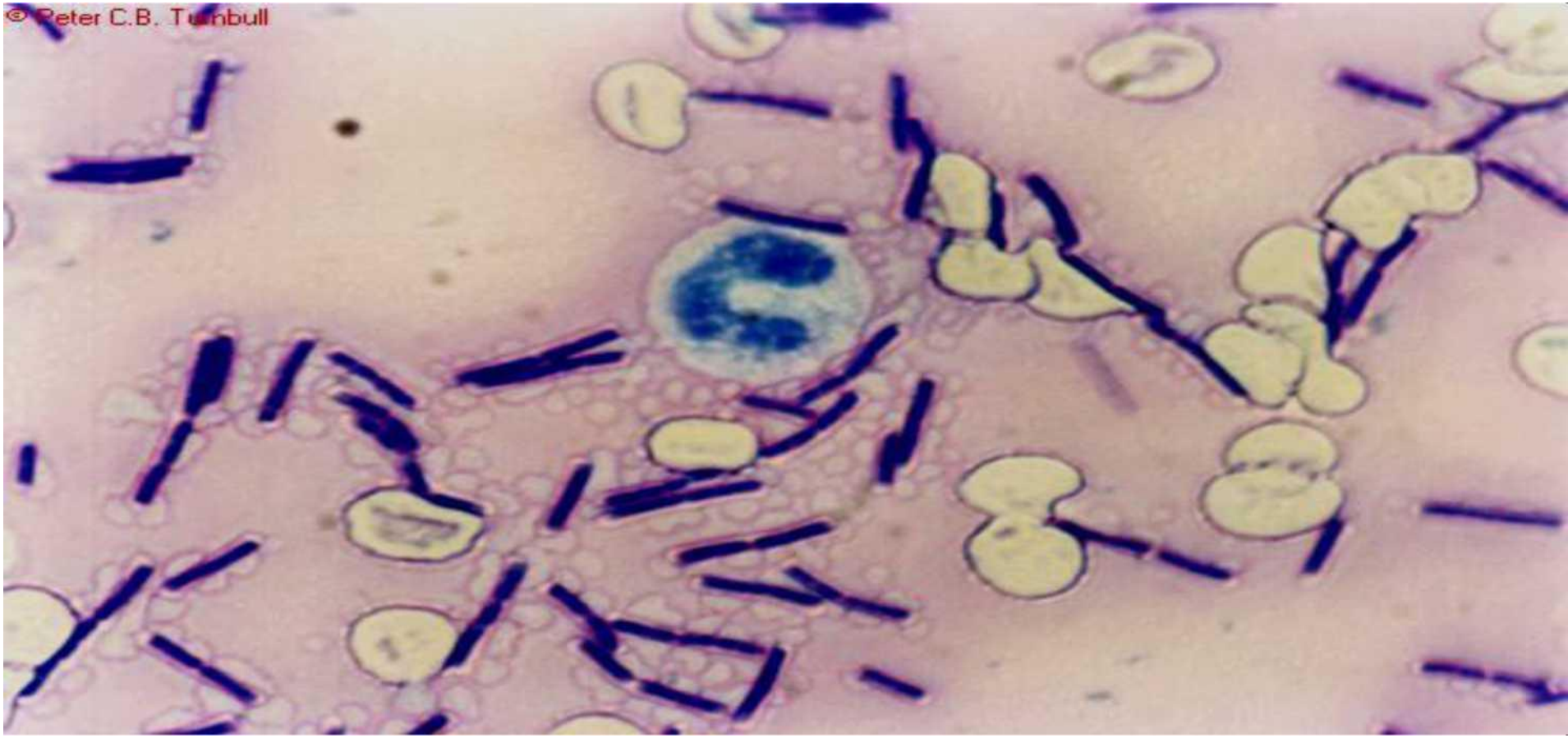
Leads to tissue damage with the formation of a chronic granuloma → cell death

Staining

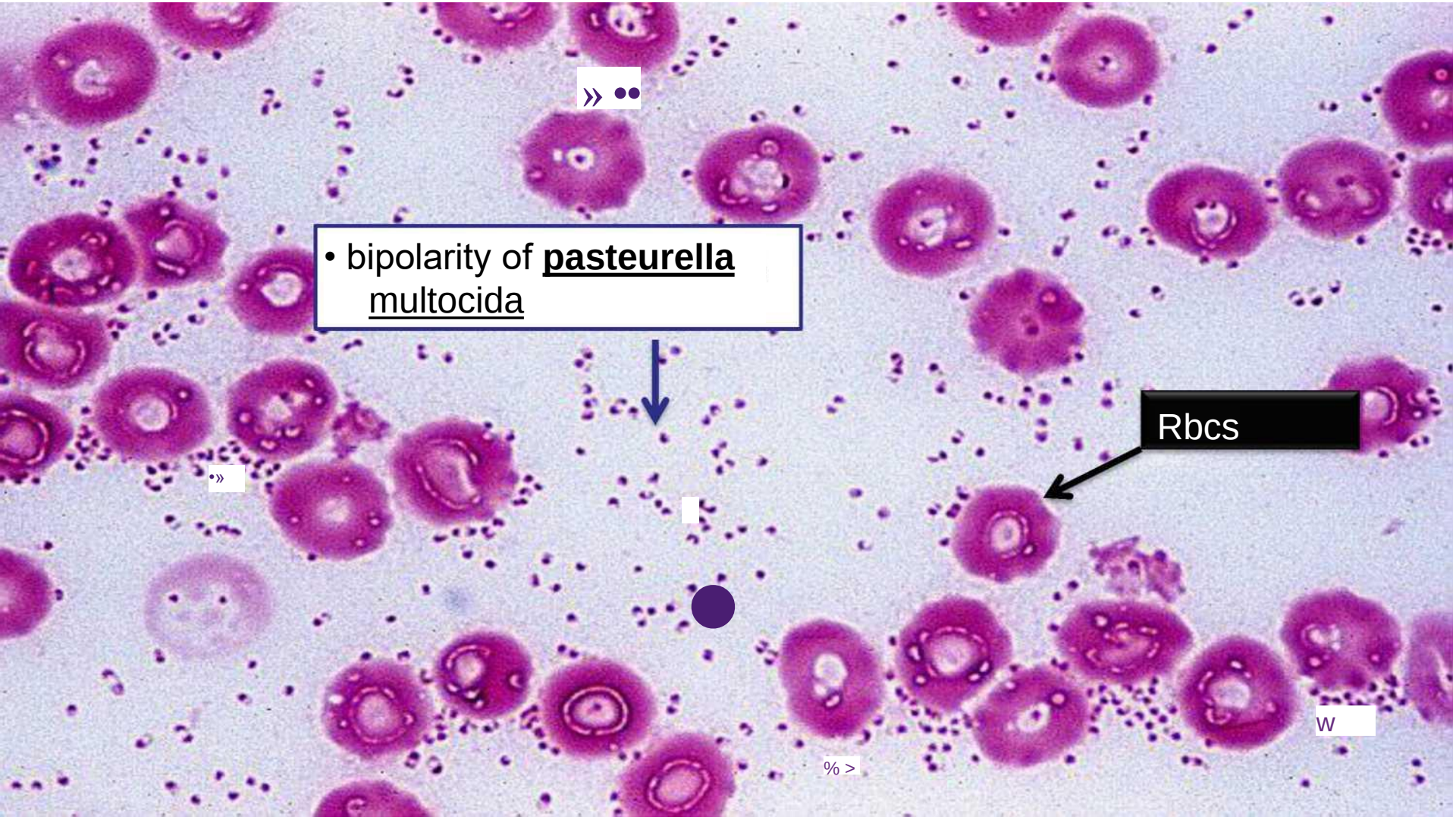


- Streptococcus agalactia
- stained with loffler's methylene blue,
- showing blue cocci arranged in short chain

- Streptococcus pyogenes
- stained with loffler's methylene blue,
- showing blue cocci arranged in shot chain

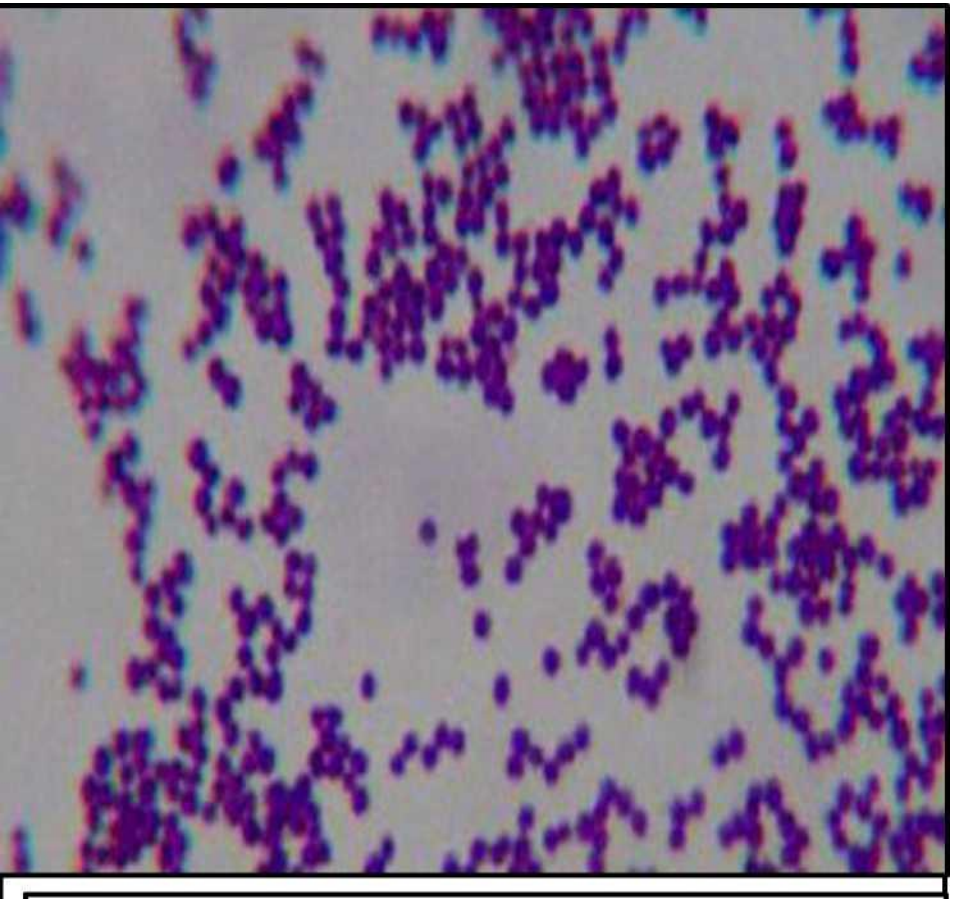


- **Blood smear from an animal that has died of anthrax(Bacillus anthracis).**
- **Stained with polychrome methylene blue,**
- **showing purple capsule and blue bacilli (MacFadyean stain).**

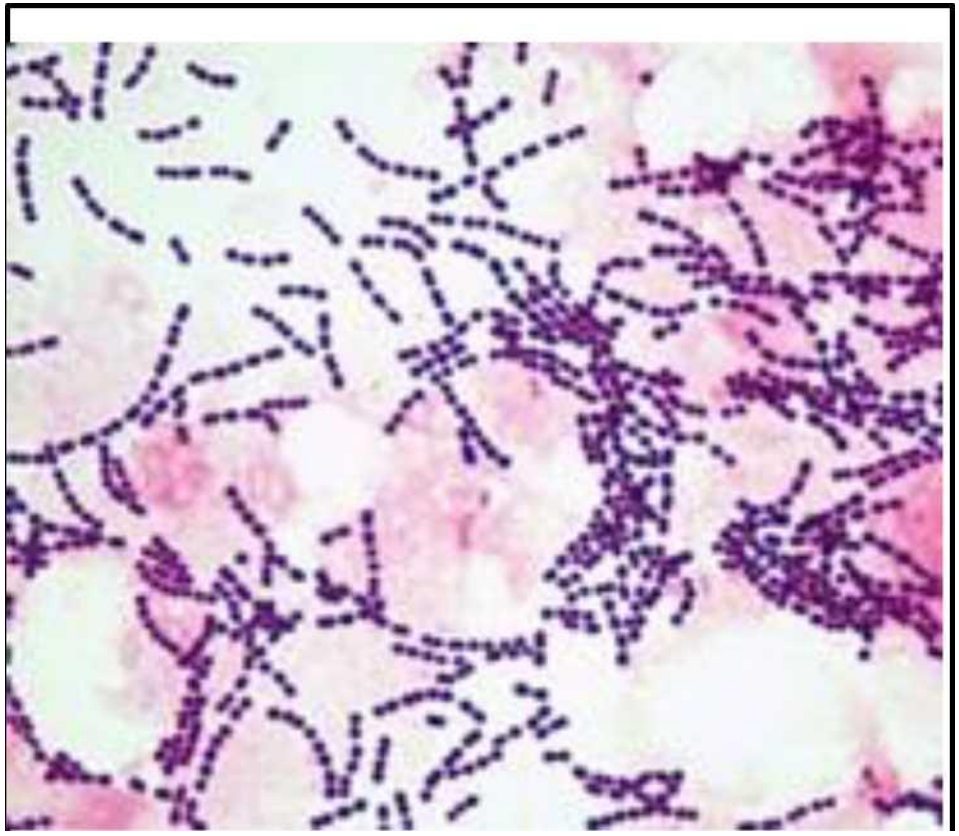


- Blood film
- stained with **leishman's stain**
- Showing pink coccobacilli WthmbjPolarity of **pasteurella multocida** 4

2- Differential staining method

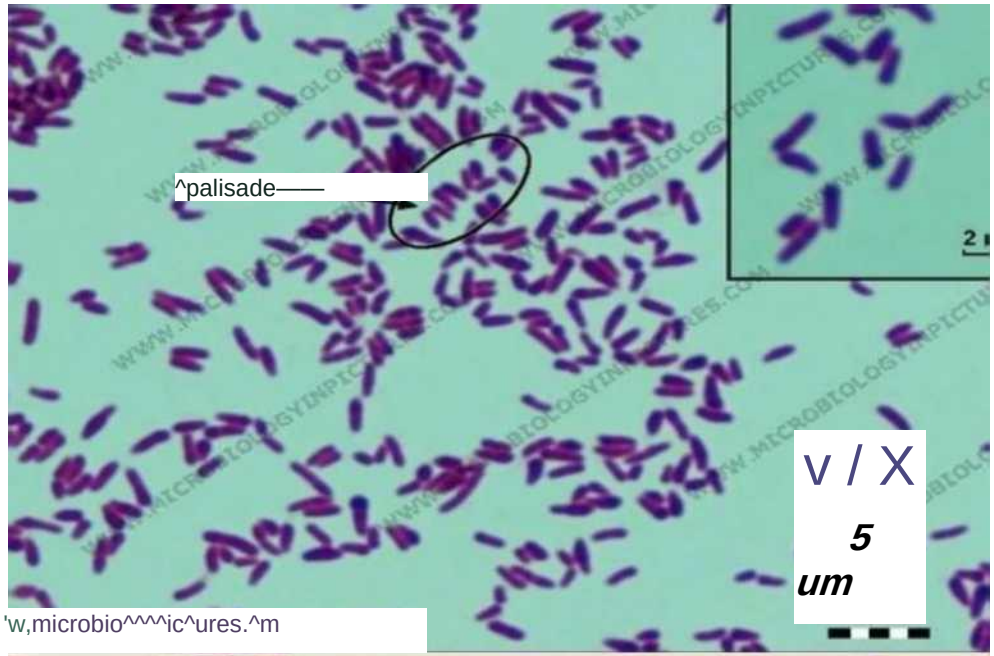


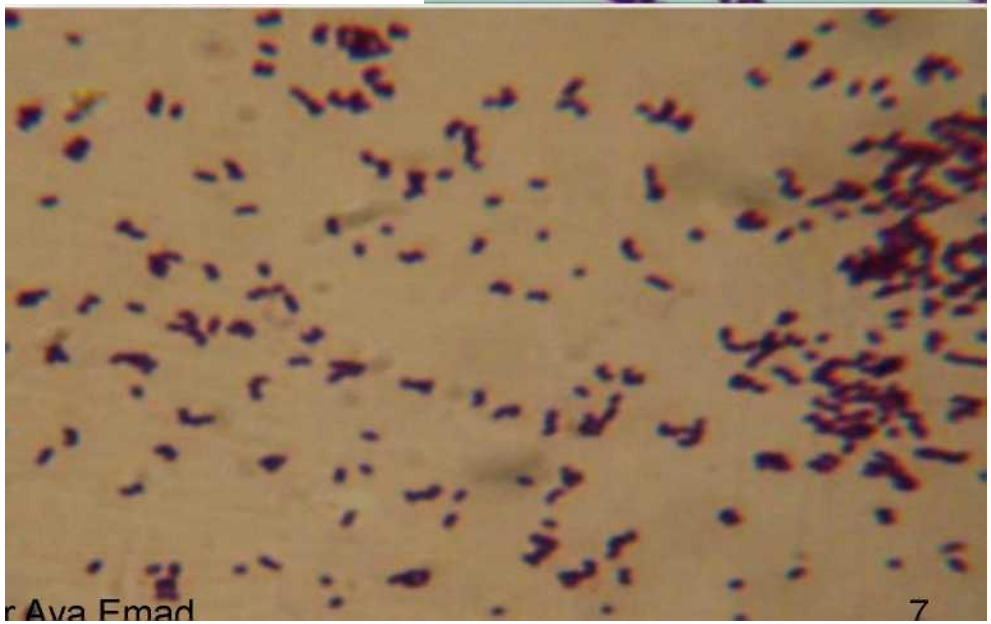
- **Staphylococci**
  - **stained with gram's stain**
  - **showing Gram positive, cocci, arranged in grape like clusters**



- **Streptococci**
- **stained with gram's stain**
- **showing gram positive cocci arranged in chain**

- Corynebacterium stained with Grams stain
- G+ve, small pleomorphic rods.
- Chinese letters shape or palisade arrangement.



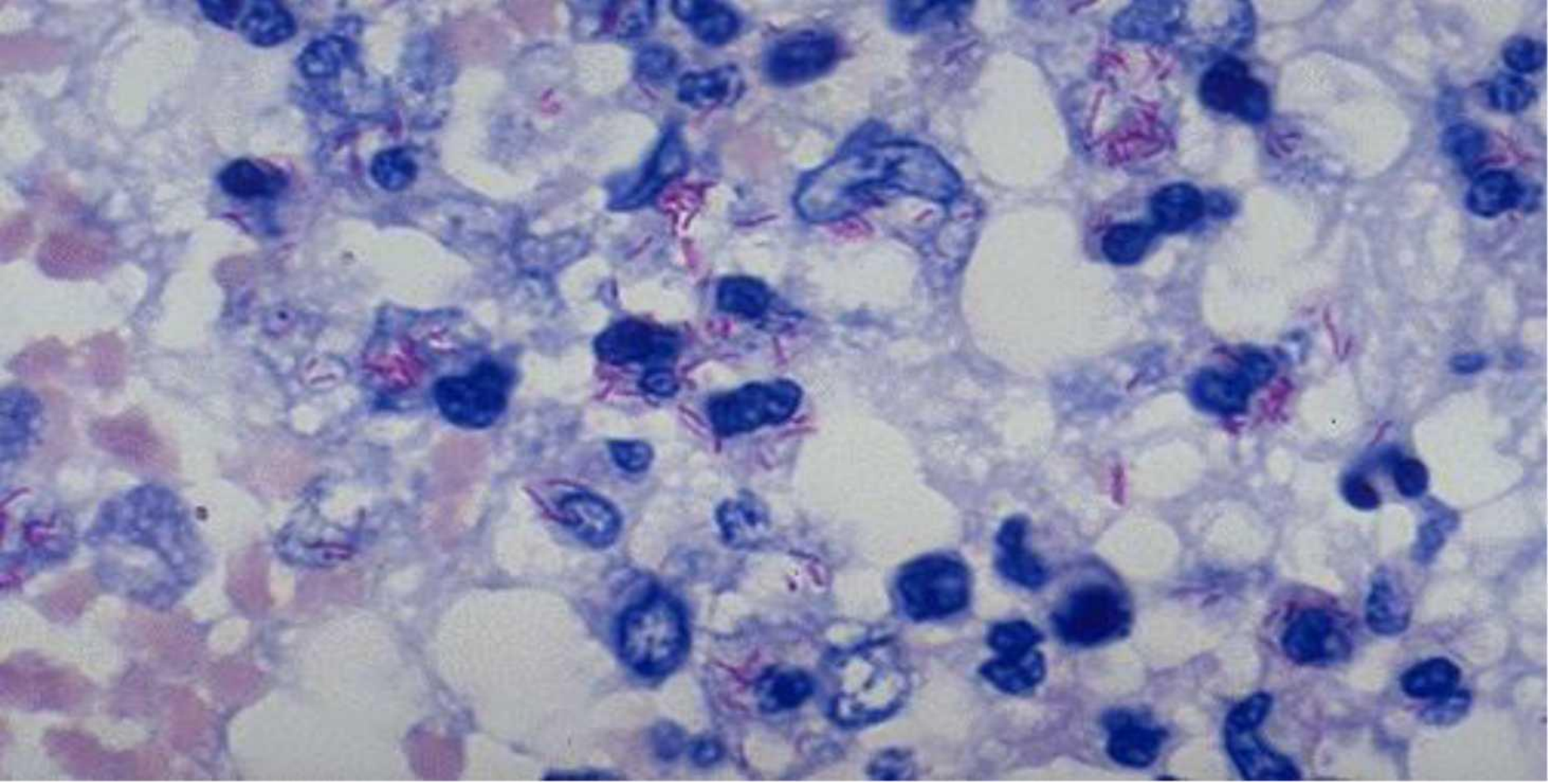


- **Clostridium tetani**
- **stained with gram's stain**
- **showing gram positive bacilli ,with drum stick appearance due to terminal endo-spore**

- ***Bacillus anthracoids***
- **stained with gram stain**
- **showing gram positive bacilli with central unstained spore**
- **arranged in long chain**

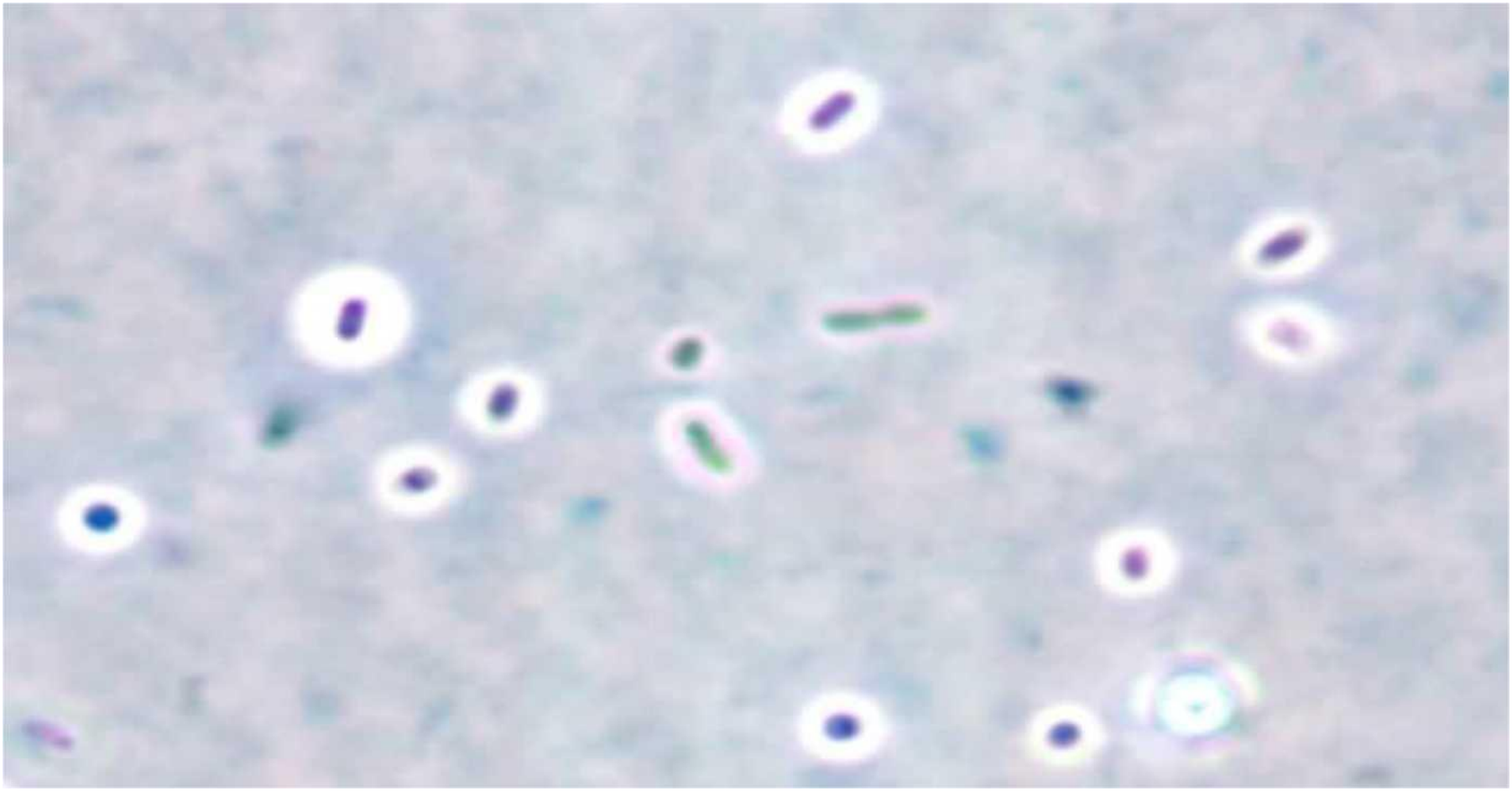
- ***Bacillus anthracis***
- **stained with malachite green stain**
- **showing red bacilli with green central spores**
- **arranged in single or pair**

- **Gram's stain**
- **showing gram negative ,cocco-bacilli (red color) ,arranged in clusters**



- ***W**cobacter/um tuberculosis*
- **stained with ziehl neelsen stain showing red long thin bacilli (acid fast bacilli) against blue backgroui**tEky**arranged in clusters**

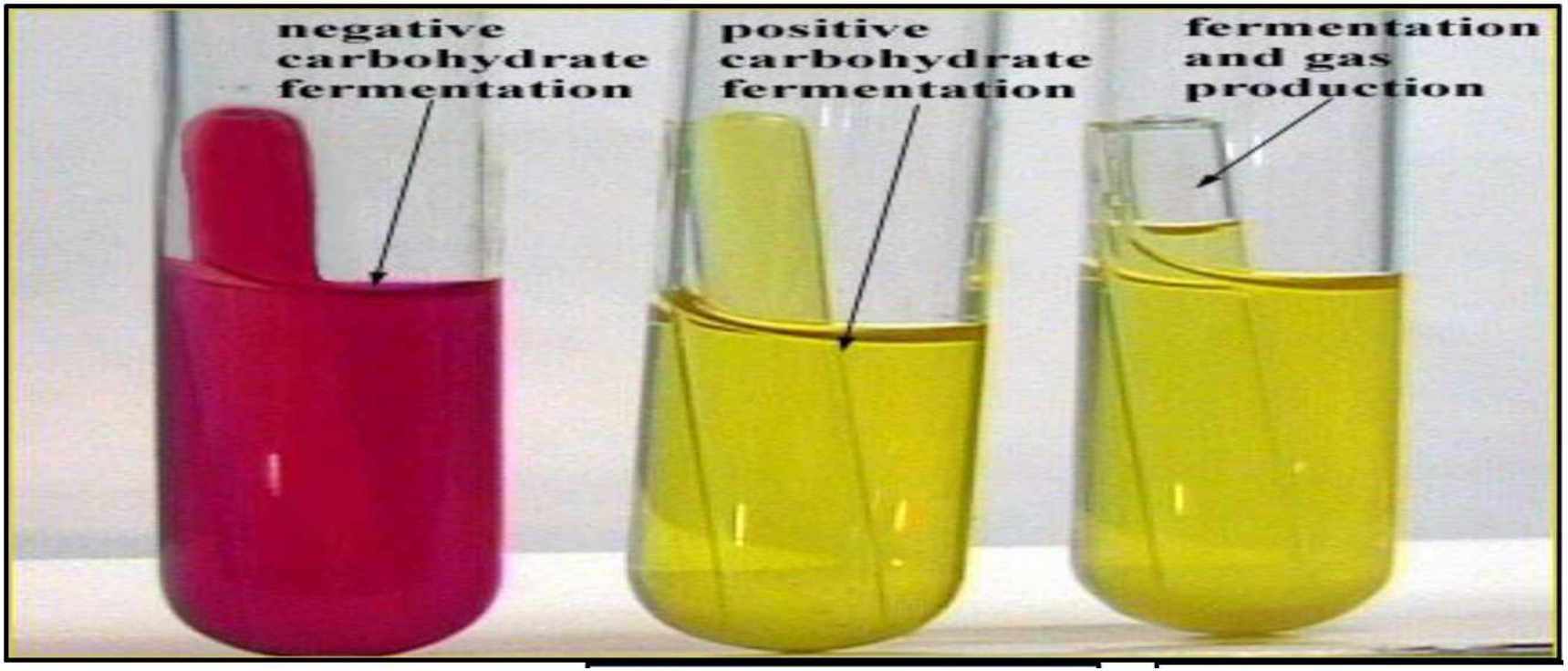
2- specific staining method



- **Dry India ink preparation**
- **showing capsulated bacteria**
- **(capsule appear as colorless zone around the blue bacterial body and agaDifis fmadue back ground)**

3-Biochemical test

Sugar fermentation test showing,



•**Red color indicate**

-ve sugar fermentation test

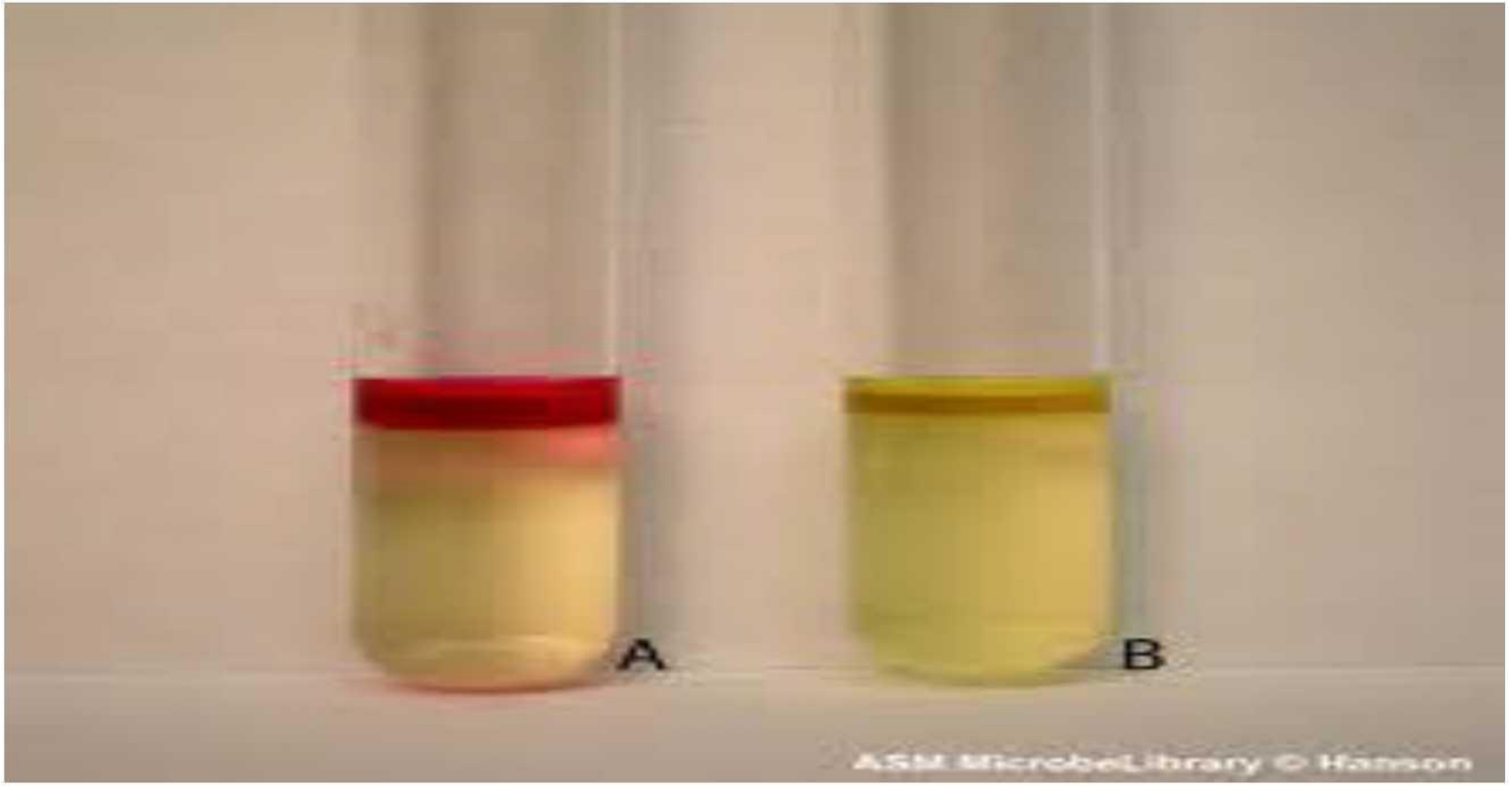
•**-ve gas production**

•**Yellow color without presence of gases in durham's tube**

•**indicate +ve result fermentation without gas production)**

•**Yellow color with presence of gases in Durham's tube**

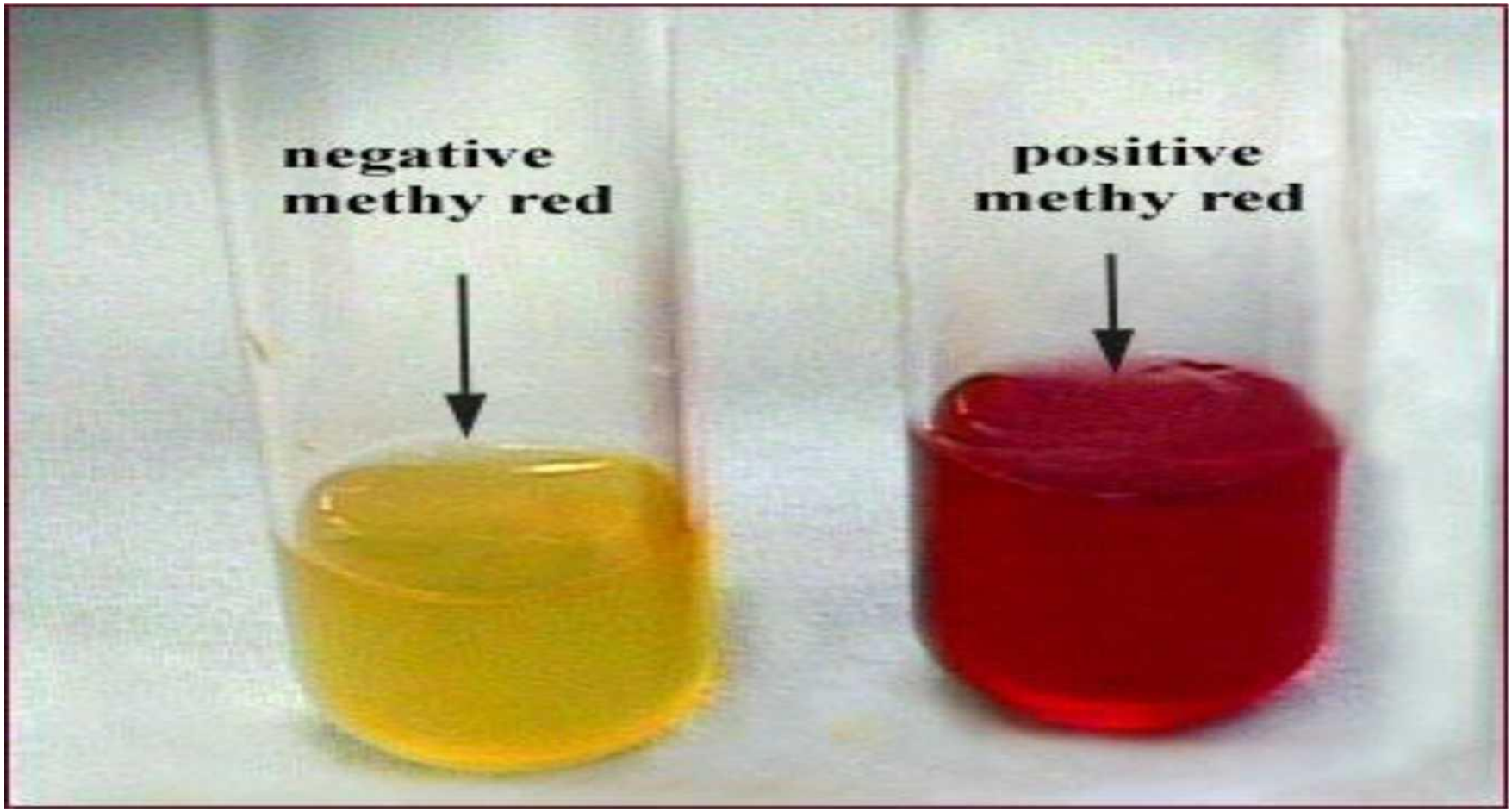
•**indicate +ve result (fermentation with gas production)**



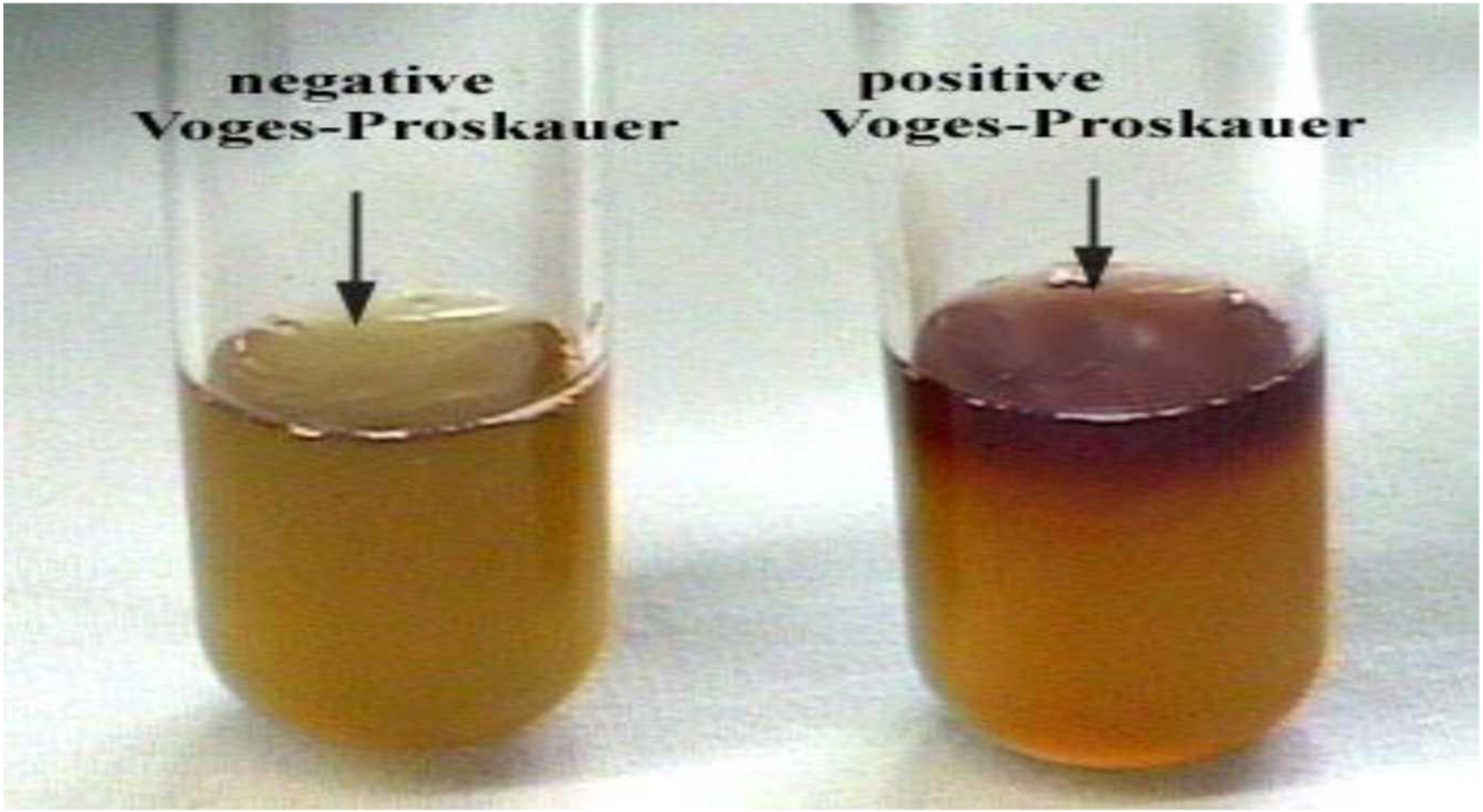
Indole production test showing from left to right

red ring: positive result

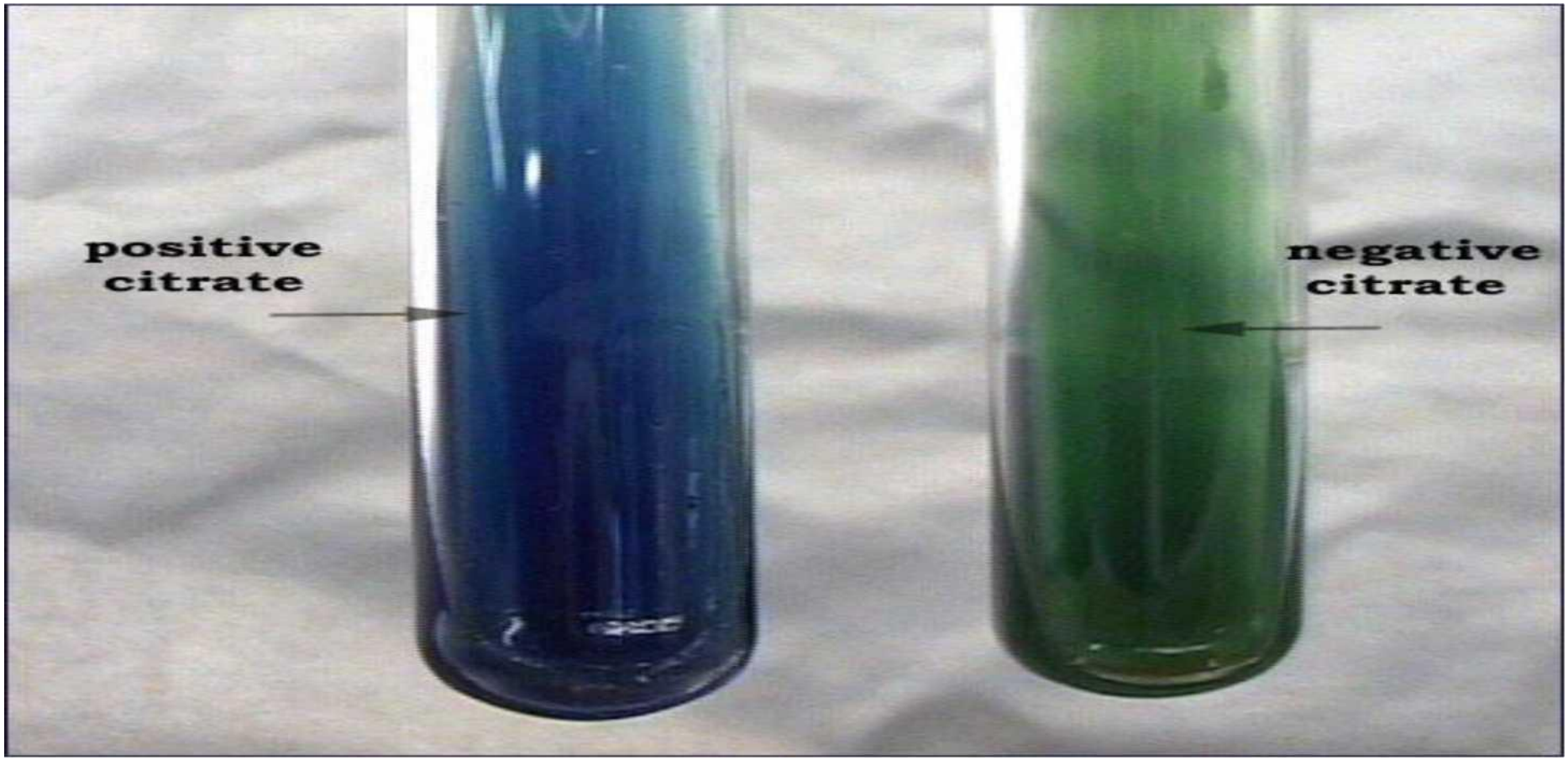
no red ring: negative result



- **Methyl red test showing from left to right**
- **Yellow color indicate negative result**
- **Red color indicative result**



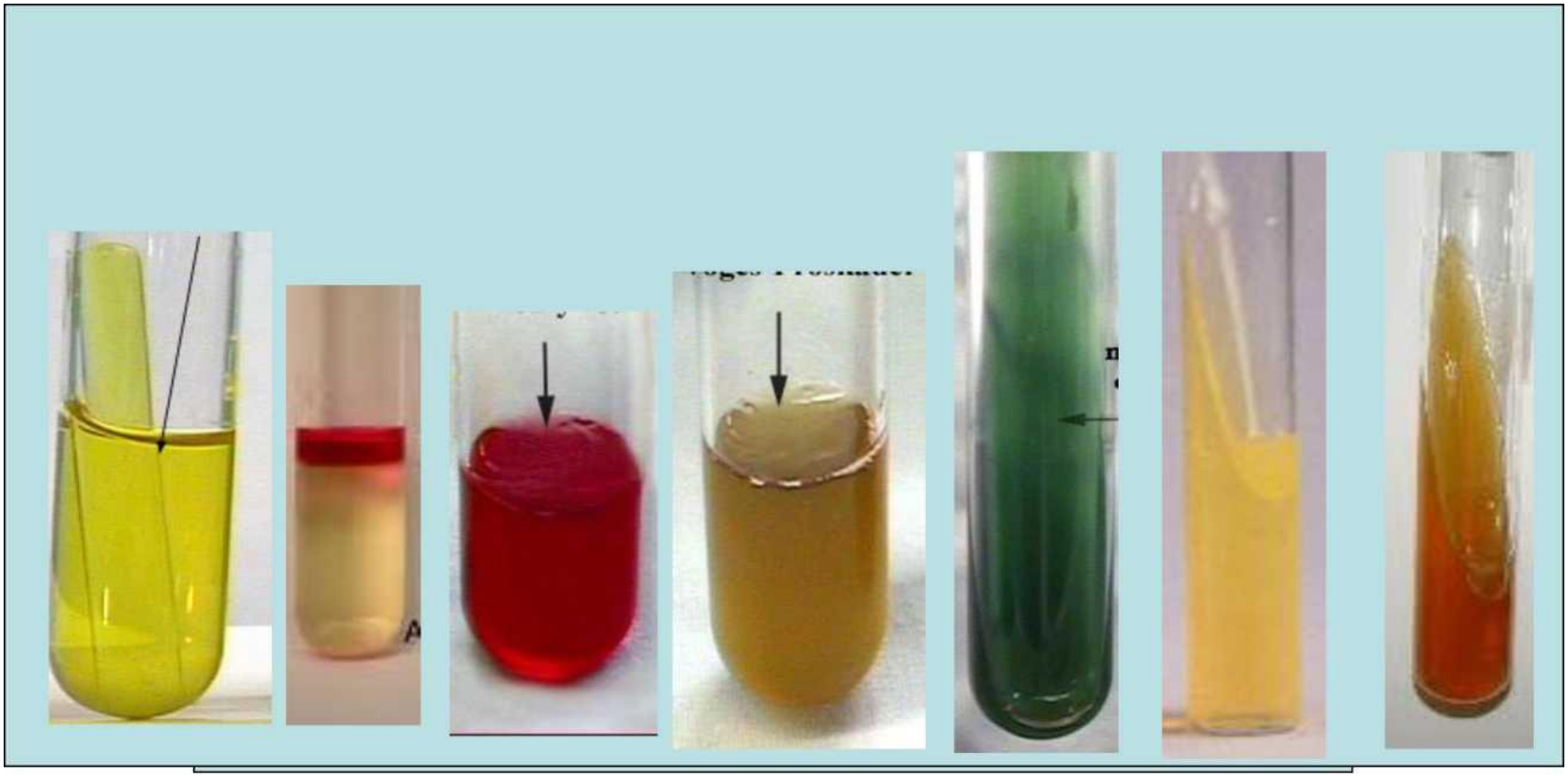
- **Voges proskauer test showing from left to right**
  - Yellow to copper color indicate negative result
  - Dark red color indicate positive result



- **Citrate utilization test showing from left to right**
- **blue color indicate positive result**
- **green color indicate negative result**

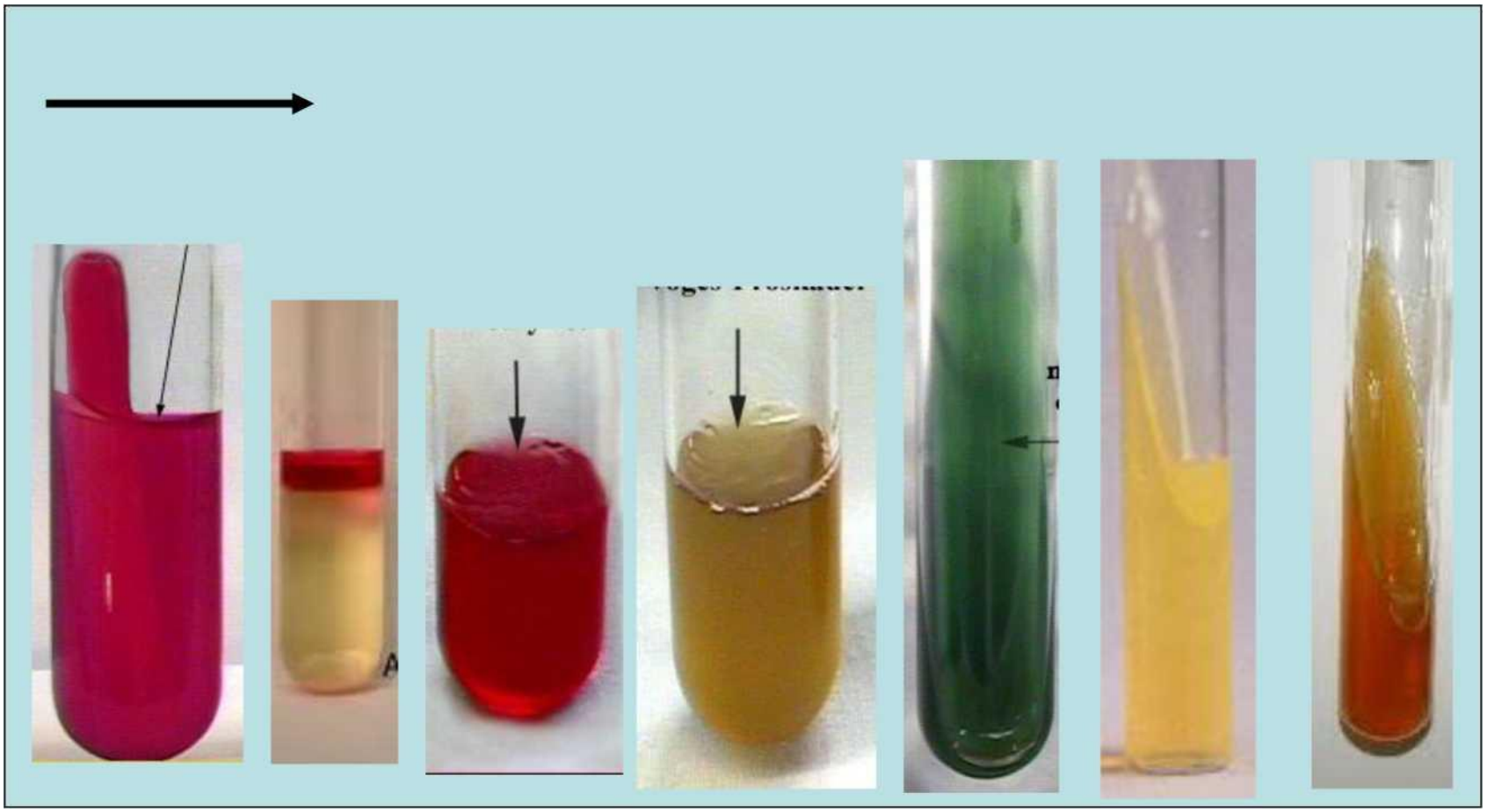
- **H_2S production test showing from left to right**
- **Black color indicate +ve result**
- **No black color indicate -ve result**

- **Urea hydro lysis test showing from left *to* right**
- **pink (foshia) color indicate positive result**
  indicate negative result



Sugar fermentation: +ve indole : +ve MR: +ve VP:-ve H_2S: -ve
citrate : -ve urea: -ve

The tested microrganism is *E.coli*



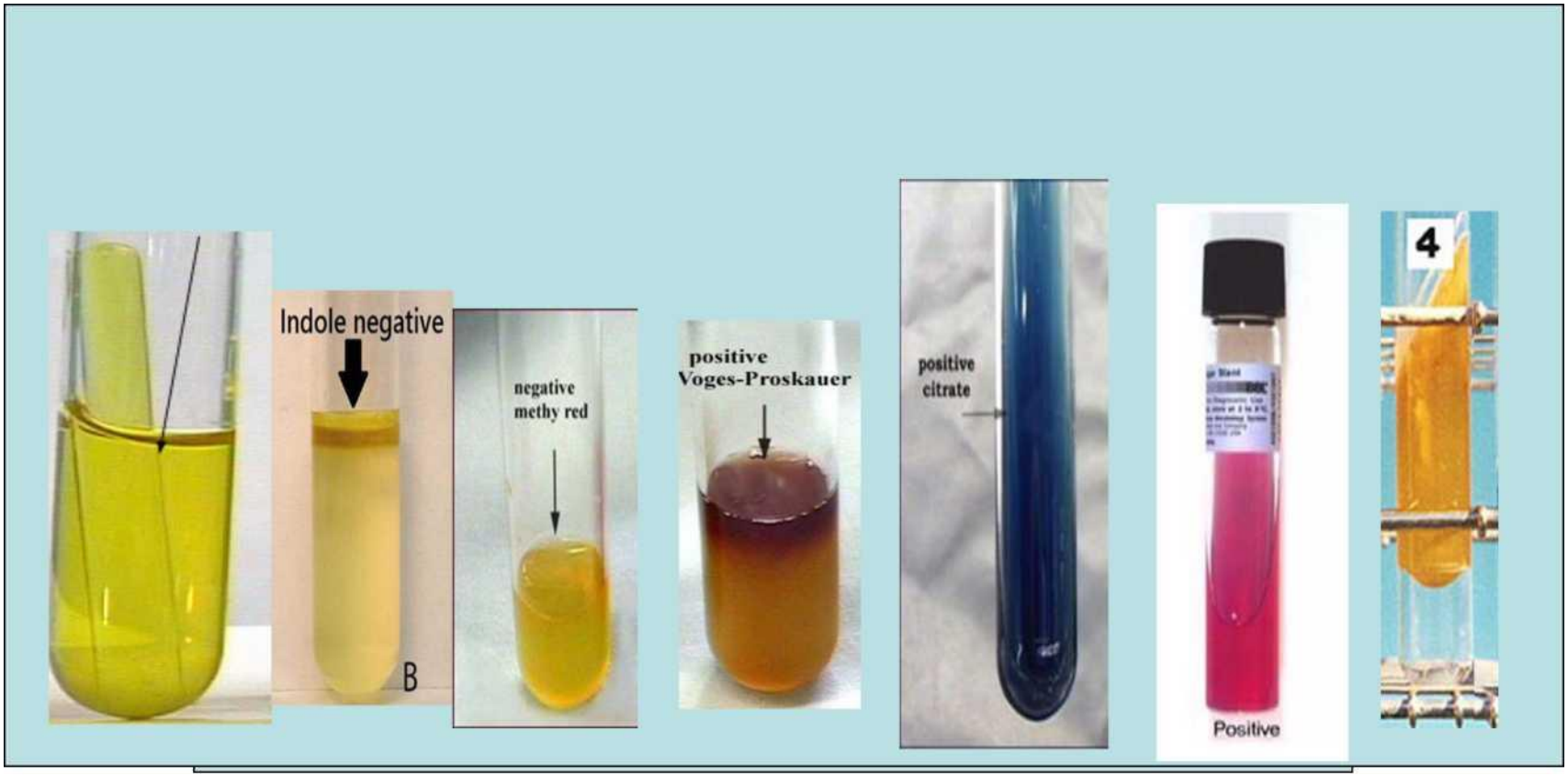
Sugar fermentation: -ve **indole : +ve** **MR: +ve**

VP:-ve **citrate : -ve** **urea: -ve**

H_2S: -ve

The tested microrganism is *shige.* *Dr.Aya* Emad

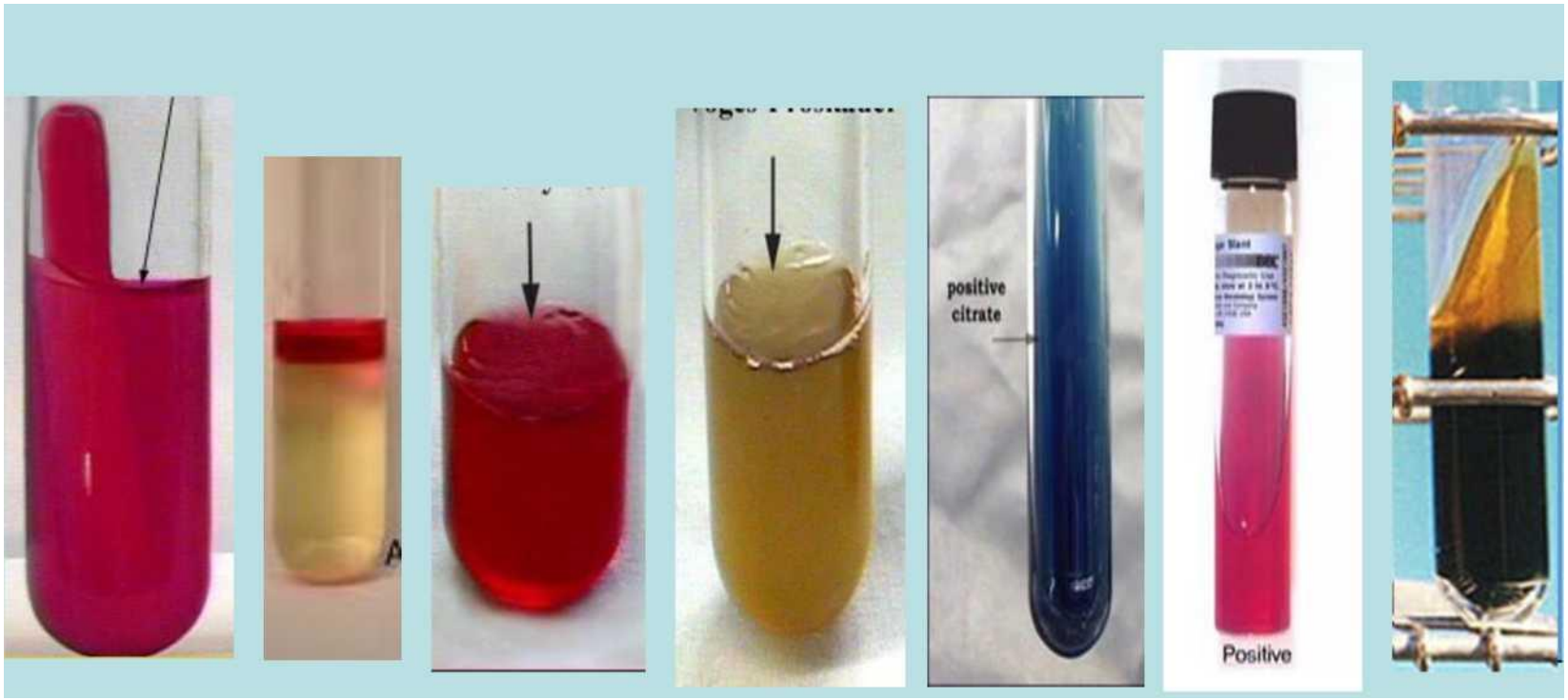
m.o.	S.F.	I	M.R.	V.P.	C.	U	H_2S
- E. coli.	+	+	+	−	−	−	−
- Citrobacter	+	−	+	−	+	−	+
- Klebsiella	+	−	−	+	+	+	−
- Enterobacter	+	−	−	+	+	−	−
- Shigella.	−	+	+	−	−	−	−
- Salmonella	−	−	+	−	+	−	+
- Proteus.	−	+	+	−	+	+	+
- Yersinia.	−	−	+	−	−	−	−



Sugar fermentation: +ve **indole : -ve** **MR: -ve** **VP:+ve**

citrate : +ve **urea: +ve** **H_2S: -ve**

The tested microrganism is klebsiella



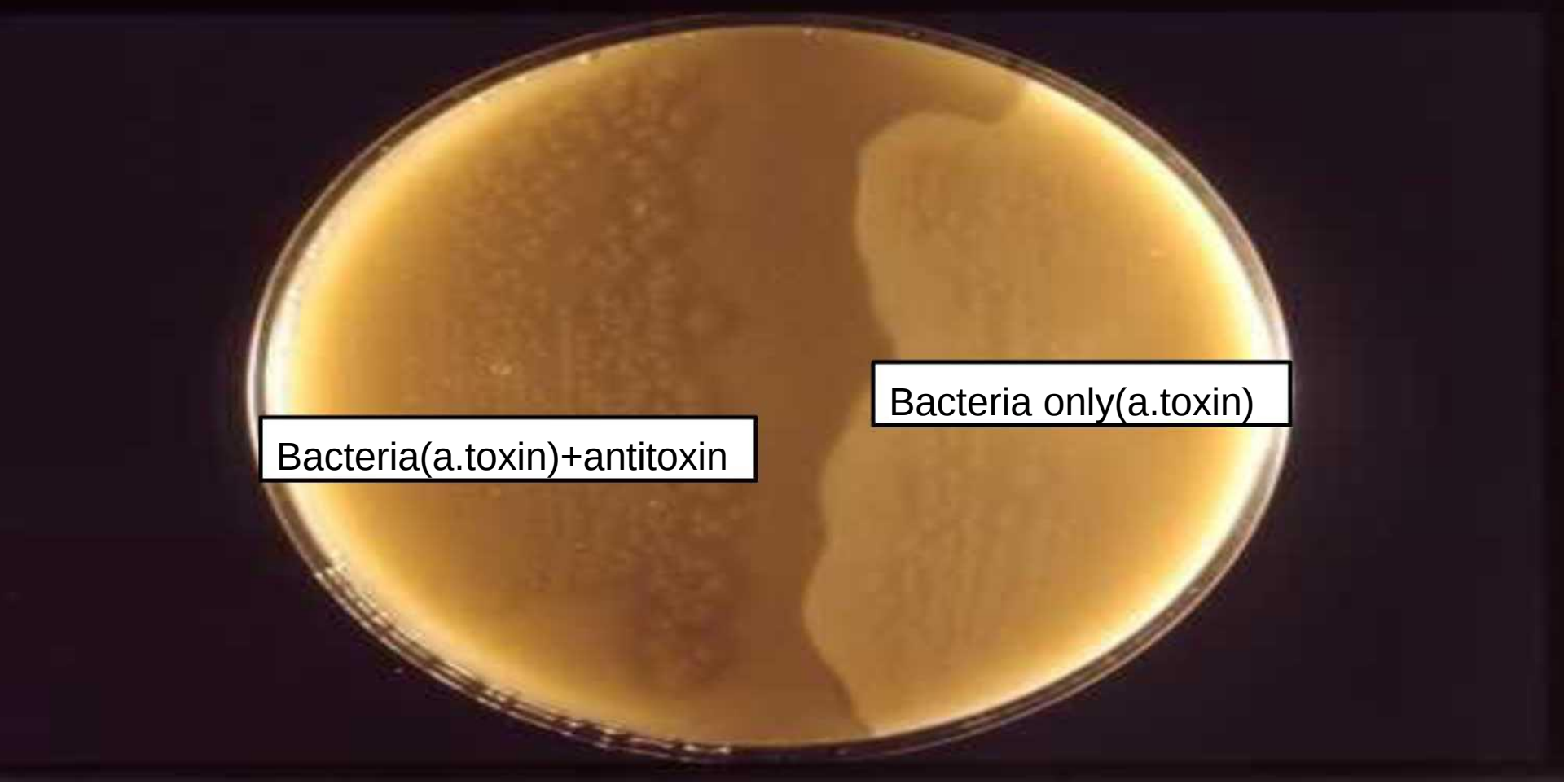
Sugar fermentation: -ve **indole : +ve** **MR: +ve**

VP:-ve **citrate : +ve** **urea : +ve**

H_2S: +ve

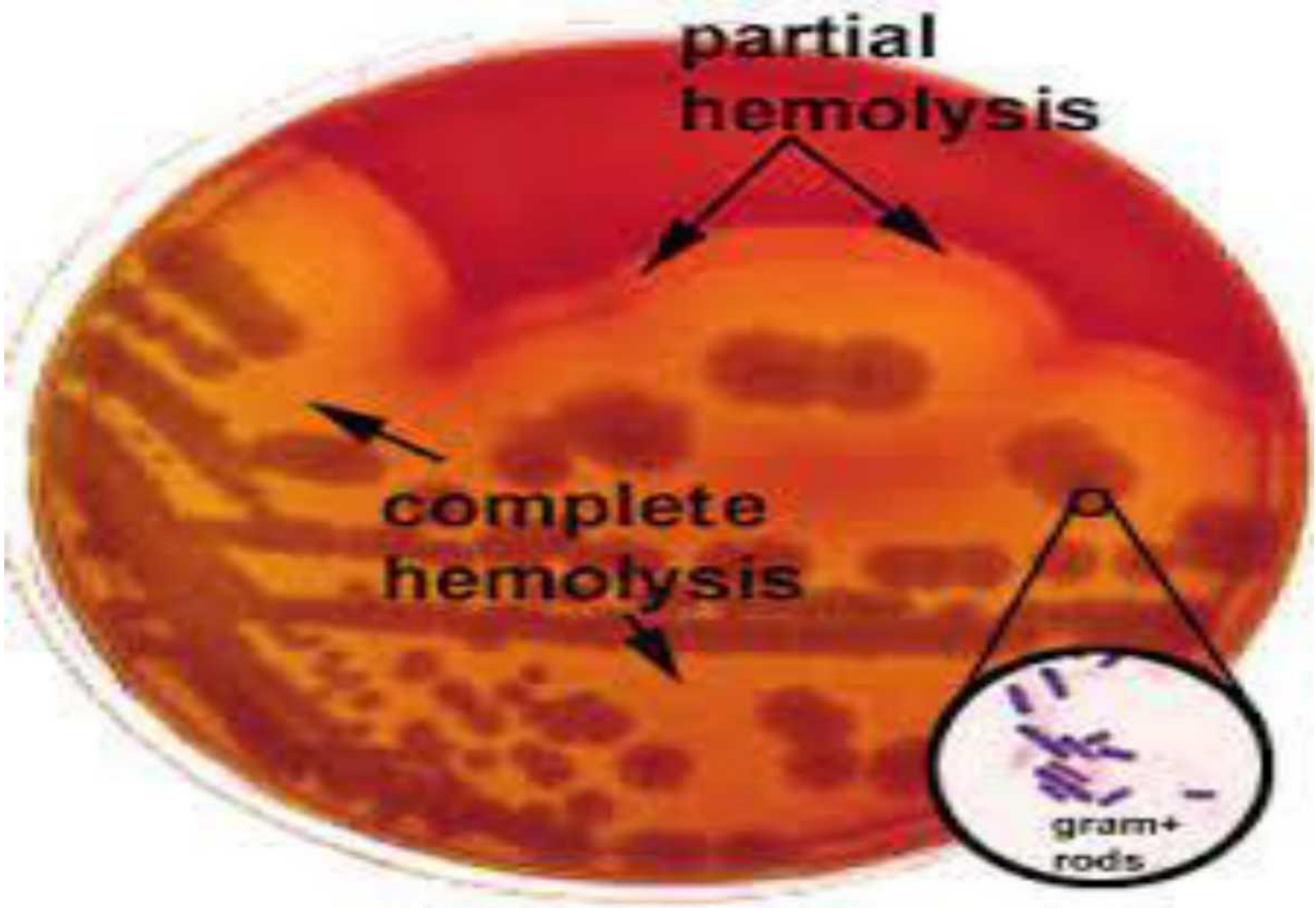
The tested microrganism is proteu^ya Emad

4- Culture ch.ch

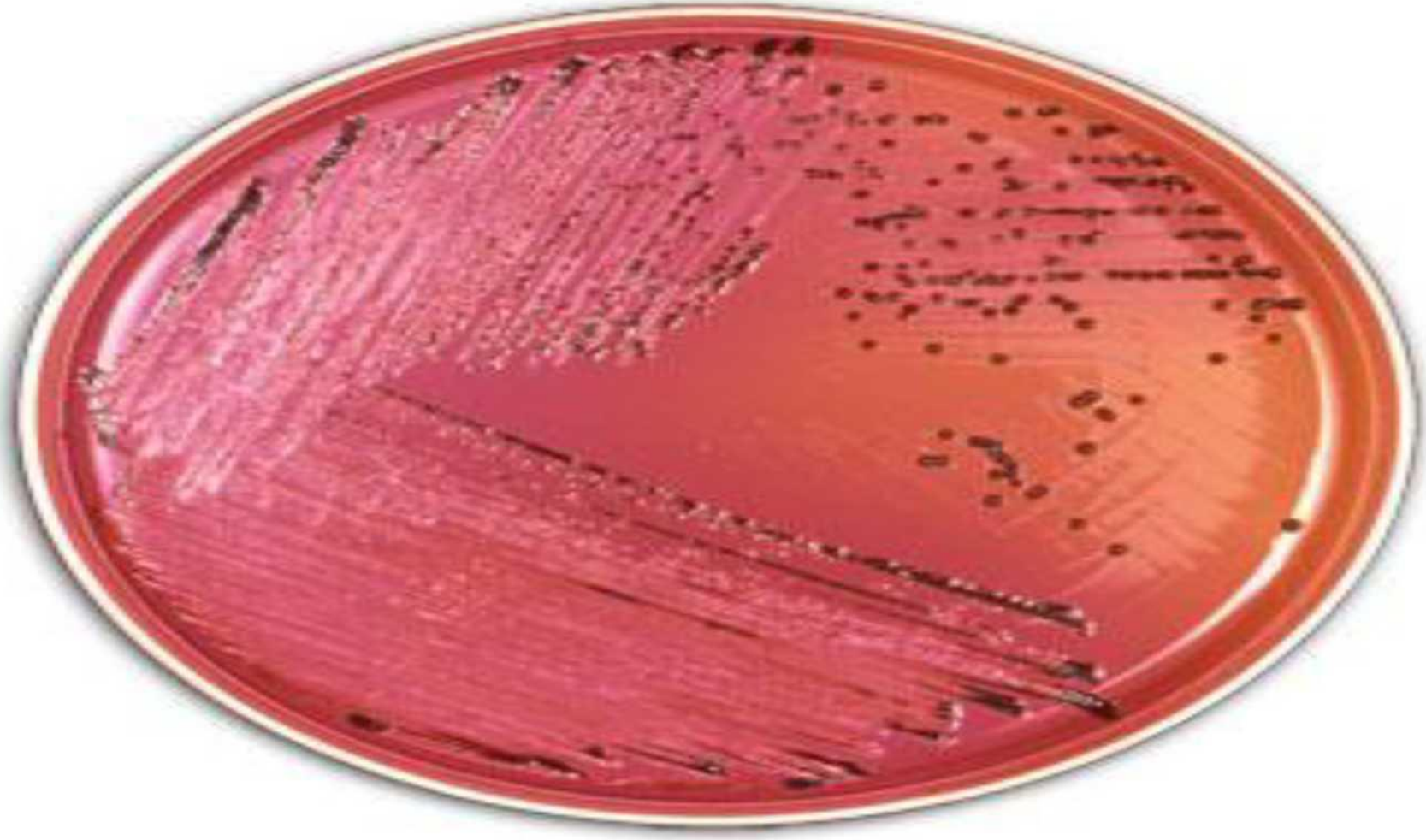


- **Egg yolk media cultivated with Cl. Welchii (perfrengens)**
- **showing opalescence due to lecithinase activity**
  - **(a toxin)**

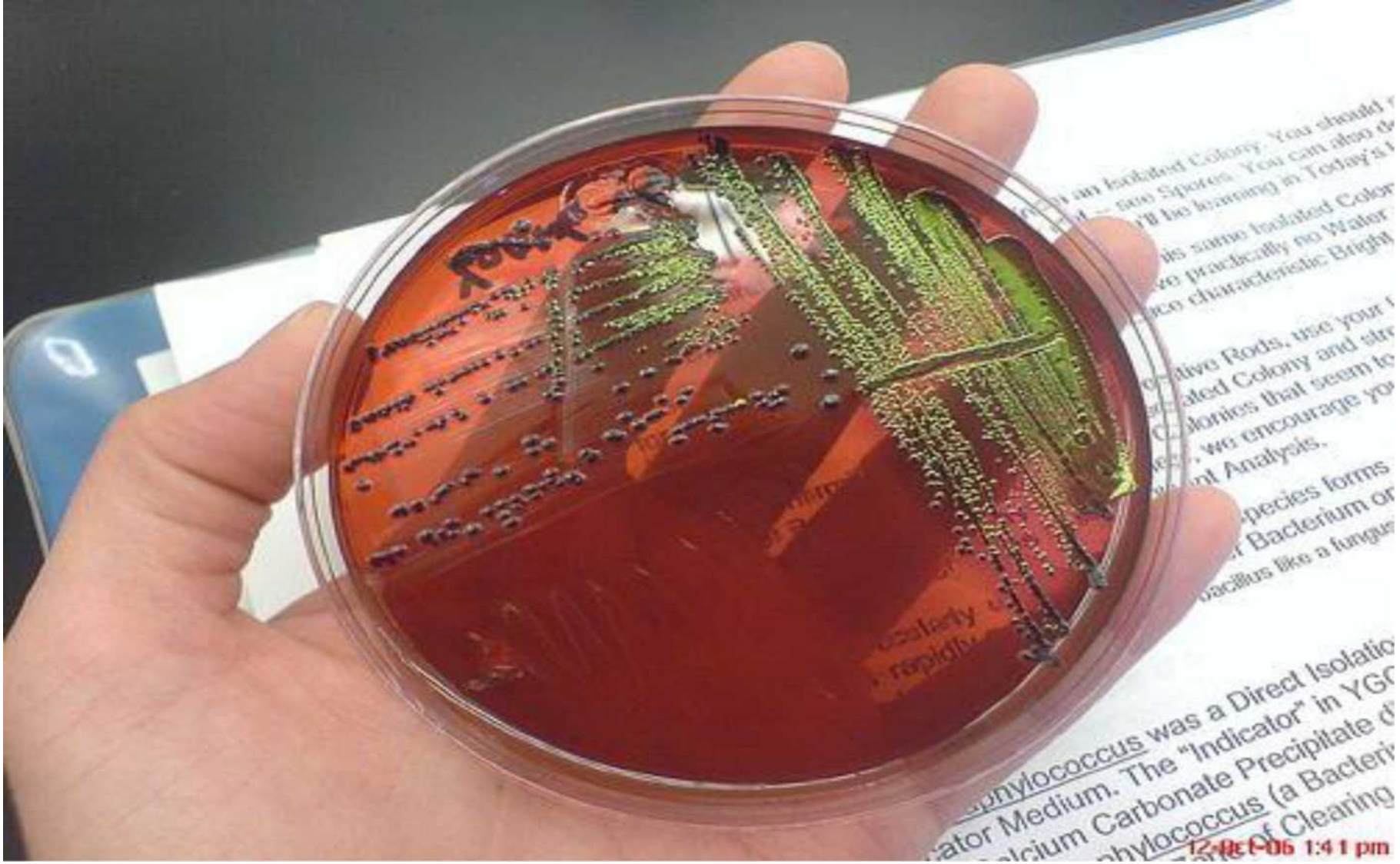
(positive Nagler reaction)



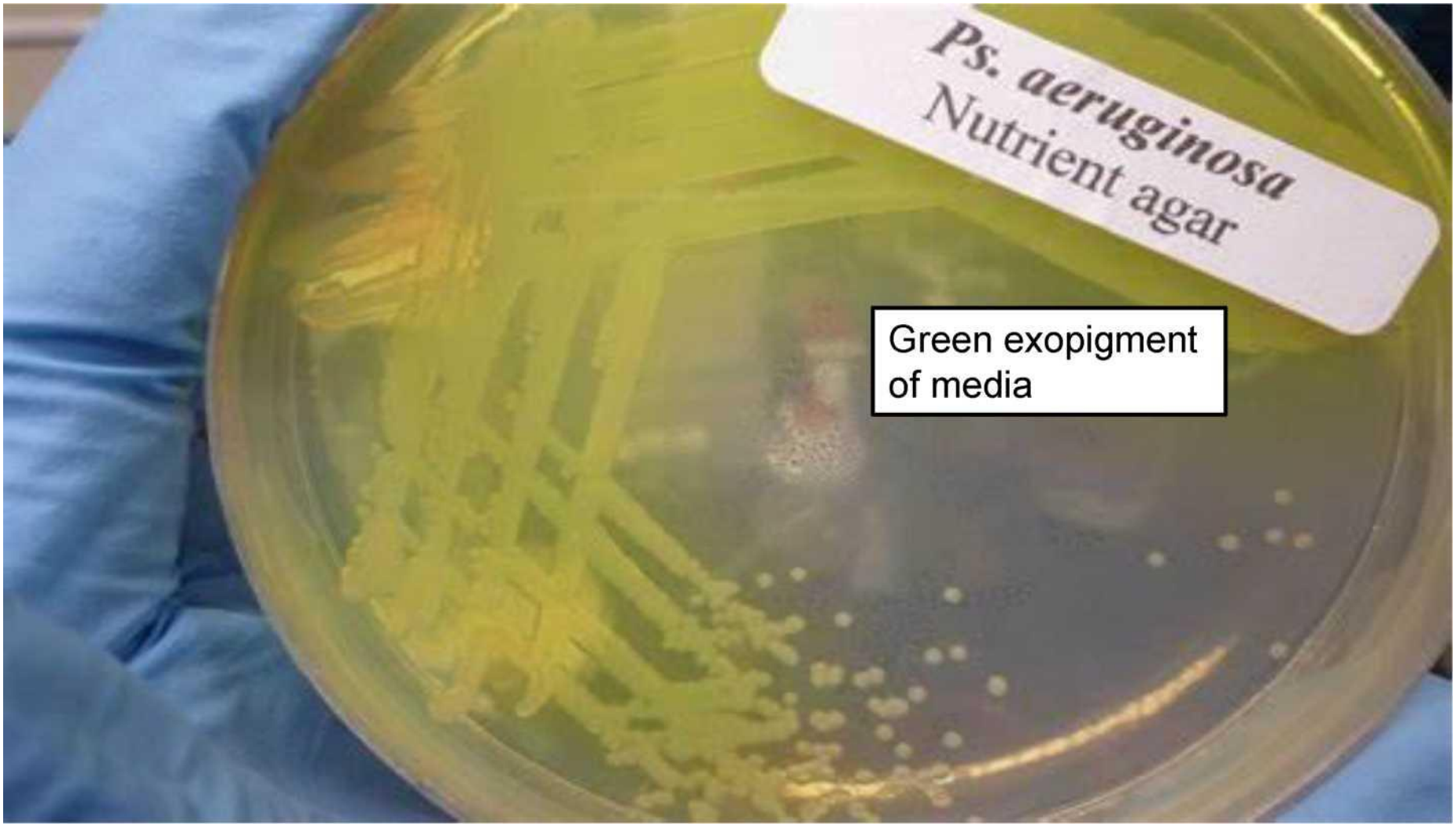
cl.Perfringens with characteristic target double hemolysis

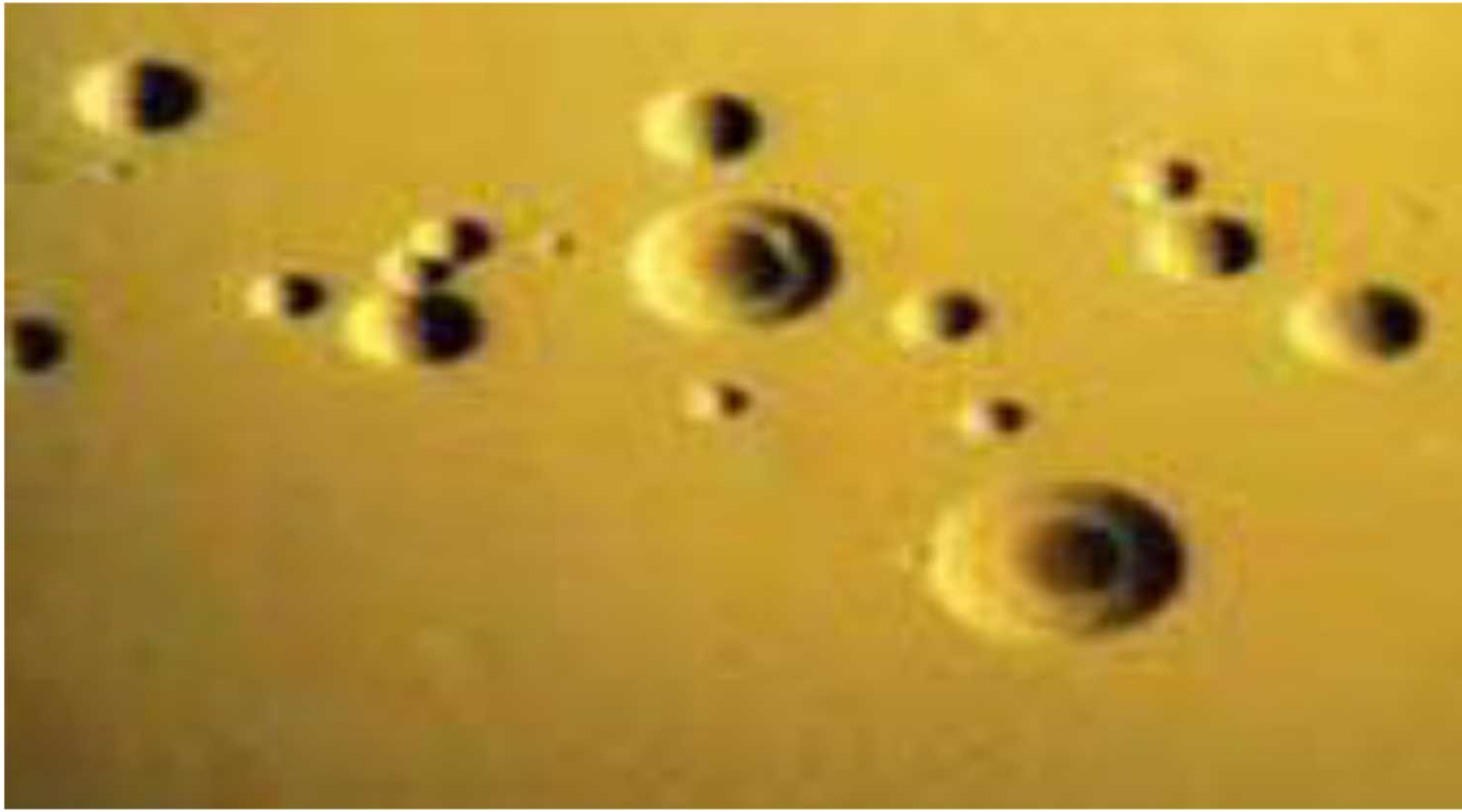


- XLD(Xylose Lysine Deoxychoiate)media cultivated with Salmonella
- showing red colony with black center(**H2s** production)



- **EMB agar (eosin methylene blue) cultivated with <u>E.coli</u>**
- **showing metalic green colony)**


Ps. aeruginosa
Nutrient agar
Green exopigment
of media



- **Microscopic colonies of <u>mycoplasma</u>**
- **which appeared as frayed egg appearance**

5- serology

Elek's test

Uses: identification of **toxigenic strain** **coty nebacterium dephtheriae**.

No precipitation line —>- result
(non toxogenic strain of corynebacterium dephtriae)

precipitation line between filter paper and cultivated line—+ve result (**toxigenic strain of cotynebacterium dephtheriae** j

Tube ring test or ascoli test result

- Use for Diagnosis of anthrax disease (bacillus anthracis).
- **+ve result** Formation of **white ring** between two solution **after 15 minutes**

Genus : Brucella

- Species :
- Br.abortus
- Br.melitensis
- Br.suis
- Br.ovis
- Br.canis
- Br.neotoma

Genus : Brucella

- Species :
- Br.abortus
- Br.melitensis
- Br.suis
- Br.ovis
- Br.canis
- Br.neotoma

Morphology & staining

- **Gram –ve**
- **Acid fast coccobacilli**
- **Singly arranged**
- **In old culture short chains**
- **Motile**
- **Non capsulated**
- **Non sporulated**
- **In tissues, occurs in groups & it is intracellular**

staining

- **Modified Zeil –Neelsen stain**

decolorization with 0.5% acetic acid

- **Koster´s stain**

depends on : brucella is alkaline fast

brucella appear as orange red against blue background

Cultural characters

- **Aerobic M.O**
- **Br.abortus need 5-10% CO2 for primary isolation**
- **Special media are needed with antibiotic inhibitory substances as:**
- **- albimi agar with ethyl violet**
- **- tryptose media with actidione**
- **-brucella agar with polymyxin**
- **-liver infusion serum agar**

- **Requires 3-4 days incubation**
- **Colonies after 2-3 days small 0.5 mm ,round,convex, translucent with small glistening surface (smooth colonies)**

- **Rough colonies are flatter, large with granular appearance & pale yellow .**

- **In tryptose broth :**

  turbidity develops slowly + fine granular deposit which become viscous after prolonged incubation

Biochemical characters

species	oxidase	H2S production	Nitrate reduction	Urea hydrolysis
Br.abortus	**+**	**+**	**+**	**+**
Br.melitensis	**+**	**Neg**	**+**	**+**
Br.suis	**+**	**Neg**	**+**	**+**
Br.ovis	**Neg**	**Neg**	**Neg**	**Neg**
Br.neotoma	**Neg.**	**+**	**+**	**+**
Br.canis	**+**	**Neg**	**+**	**Neg**

Antigenic structure

- **Smooth strains posses 2 antigens**
  A & M
  A:M ratio (quentative difference)

By aggl.absorption test using monospecific A or M antisera

Used with other tests to differentiate 3 main species:

Br.abortus **20 : 1**

Br.melitenesis **1 :20**

Br.suis **1 : 1**

Symptoms & lesions

- **Human → milk ,meat ,contact or M.M**

  malta fever, undulant fever or mediternian fever

Cows → contagious abortion or Bang´s

disease abortion 7-8 month of pregnancy

virulent strains have a protective outer protein coat allowing them to survive intracellular → chronic infection

- **Bulls** can be isolated from L.nodes of the digestive tract & spleen
- No lesions recognized

- **Sheep & goats**

Br.melitenesis

- **abortion 3-4 month** of pregnancy .
- lameness & mastitis
- The udders become infected in a high percentage of cases
- **Br.meletenesis is shed in milk**

- **In bulls → epididymitis & testicles abscesses usually develops & the organs are destroyed**

Laboratory diagnosis

- 1- history 2-samples

source	types	examinations
Aborted fetus	Stomach content Intestinal content Lung tissue. spleen	Direct smear bacteriology
Mother after abortion	Uterine discharge Fetal fluid	Direct smears, bacteriology G-pig inoculation
2-3 weeks after abortion	Blood Milk whey	Serological : Abortus bang test Tube aggl.test
bulls	Preputial wash Semen serum	G,pig inoculation Bacteriology Serological test

- **3- direct smears:**

Fresh samples of chronic tissues

Fetal stomach content

Uterine discharge

Films stained by the differential stains for brucella

Brucella occurs characteristically in clumps intracellular as well as extracellular

Bacteriological examination

- **Isolation of brucella**

On the special media containing antibiotic

Incubated 37ºCfor 10 days at least

Br.abortus & melitenesis required 5-10% CO2

For primary isolation

- **Differentiation of the isolates**

tests	Br.abortus	Br.melitenesis	Br.suis
CO2 requirment	+	-	-
H2S production	+	-	-
Urease hydrolysis	+ (1-2 hr)	variable	+ (0-5 hr)

Characters	*Br. abortus*	*Br. meletensis*	*Br. suis*
Dye sensitivity:			
Basic fuchsin	+	+	-
Methyl violet	+	+	-
Thionin	-	+	+
Pyronin	+	+	-
Antigenicity (A:M ratio)	20:1	1:20	1:1
Agglutination by:			
Monospecific M antisera	+	+++	++
Monospecific A antisera	+++	+	++
Susceptible animals	Cattle	Sheep and goat	Swine
Virulence to man	Weak	High	moderate

- **G.pigs inoculation :**

The infected material inoculated S/C in 2 male G.pigs to obtain a pure culture of brucella

+ve → orchitis i,e strauss reaction within 3-8 weeks

The organism in pure culture can isolated from spleen ,liver & testicle

The serum of slaughtered G.pigs can be examined the presence of antibodies

Serological tests

- **For brucella diagnosis by indirect detection of antibodies in serum , vaginal mucous, milk, whey & semen –serum**

- **1-agglutination test :**
- **1.1 : serum aggl. test or tube aggl. test (SAT) :**
- **By using standard Br.abortus**
- **+ve titer = 1/40**
- **Suspected titer =1/20**
- **Accurate, simple**
- **The test repeated after one month**

- **1.2 : plate rapid method or slide agglutination test :**
- **Stained heavy suspension of Br. abortus killed antigen**
- **Applied to give screening on a herd level**
- **Serum ,whole blood . Whey may be used**
- **The result read in several min.**

- **1.2.1 : Rose Bengal test (RBT) :**

Is a type of slide agglutination

by using stained antigen with Rose-Bengal

- **The test is rapid & it give –ve reaction**

- **used to differentiate between vaccinated from infected animals**

- **2- Abortus Bang Ring test (ABRT)**
- **Or Milk Ring Test (MRT):**

1 drop of stained (heamatoxylin , blue or tetrazolium ,red) heavy suspension of killed Br.abortus + 1 ml of milk

Or 1 ml antigen + 9 l milk then mixed well

is incubated at 37ºC in water bath for 1-2 hr especially for buffaloes , sheep, goat milk

-ve	+ve
No change	decolourized milk
Bluish color	capped by bluish violet cream

Used extensively in herd level for survey purposes & general screening method

- **3. complement fixation :**

Used for detection of chronic infected animals

In case of repeated aggl. Tests give unreliable results

4. Fluorescent antibody technique:

Used for examining the highly contaminated materials such as :

retained placenta , cotyledons or vaginal discharge

Immunity & control

- **The immunity is depending upon cell mediated immunity by using attenuated vaccines**
- **The commonly used vaccines are:**

1- buck strain :

Avirulent strain of brucella to cattle

It is administered as a single S/C dose

Vaccination of calves (3-12 months) as widely used as calf hood vaccination

- **2- McEwen 45/20 vaccine (rough brucella abortus vaccine) :**

- **It is not virulent enough to cause disease in cattle**

- **The rough 45/20 does not produce agglutinins in animals (advantage over than strain 19)**

- **3- a killed 45/30 adjuvant vaccine (K45/20A):**

To avoid the disadvantages of the rough strains

Order: EUBACTERIALIS

Family: LACTOBACTERIACE

Genus: STEROTOCOCCUS

SPECIES:

Sterptococcus pyogenes

Sterptococcus agalactiae

Sterptococcus dysagalactiae

Sterptococcus uberis

Sterptococcus equi

Sterptococcus zooepidemicus

Sterptococcus canis

Sterptococcus gallinerum

General characters of Streptococci:

- It is spherical or ovoid cocci in shape 0.5 µ in diameter, it is usually arranged in chains of various length, gram positive .they are non motile, non sporulated but some strains are capsulated (slime layer).

- **<u>Growth requirements and cultural characters:</u>**
- Streptococci are aerobic or facultative anaerobic, grow weak on ordinary media but grow well on blood or serum agar at 37ºc and ph 7,2 . The organism produces haemolysis on blood agar plate the specific media are ***Edward´s blood ager*** media and sodium azide agar media.
- Streptococcus doesn't produce pigments either in fluid media or on solid media. The colonies on blood agar are small, smooth – glistening after 48 hours, while in broth granular growth and powdery sediment are formed within 36-48 hours.

- **<u>Classification:</u>**
- **1-according to type of haemolysis :**
- Streptococci causing complete haemolysis (Beta (β) haemolysis), a clear zone of haemolysis as *Strept.pyogenes* and *Strept.equi*.
- Streptococci causing in complete haemolysis (alpha α) such as *Strept.viridians* and *pneumococci*.
- C) Streptococci causing no haemolysis (gamma γ), non hemolytic streptococci as *Strept.lactis* and *Sterpt.salivaris*.

- **2-Lancifield classification:**
- Lancefield reported that the hemolytic streptococci could be divided into groups (A-N) on serological basis depending on precipitation test using the specific carbohydrate substance designated as C.substance as the antigen which differed in each group:
- - Group A: *Streptococcus pyogenes*
- - Group B: *Streptococcus agalactia*
- -Group C: animal streptococci as *streptococcus zooepidemicus*, *Streptococcus dysgalactia, Streptococcus equi, Streptococcus canis.*
- -Group D: Enterococci as streptococcus fecalis ,*Streptococcus faecium*
- -Group E: Strains isolated from milk and dairy products as *Streptococcus uberis*
- -Group F: Strains isolated from respiratory tract.
- -Group G: Strain isolated from respiratory tract of man and dogs.
- -Group H, K, M and group N

- **3-Sharman classification:**
- It depends on the biochemical characters of the enterococci or non pathogenic streptococci on the following reactions:
- Growth at 10 ºc and 45ºc as *Strept.viridians*.
- Tolerance to 6.5 % sodium chloride.
- Reduction of methylene blue.

- ***Streptococcus pyogenes***
- **Morophological characters:**
- *Strept.pyogenes* are arranged in long chain, it is cocci , non motile , non sporulated, has capsule in young culture and gram positive
- **Distribution:**
- It is widely distributed on the skin and in the mucous membrane

- **Growth requirements and cultural characters**:
- *Strept.pyogenes* grow on media containing blood or serum at 37ºc and PH 7.2 .
- Its colonies on the solid media are very small, smooth glistening, dew drop like, finally granular and not pigmented.
- The colonies produce zone of complete haemolysis (beta haemolysis)on blood agar after (24-48) incubation

- **Biochemical properties:**
- The organism ferments glucose, lactose,sucrose, and salicin producing acid only do not ferment sorbitol, do not liquefy gelatin, catalase negative and indol negative.
- **Resistance:**
- *Strept. pyogenes* killed by 60ºc for half hour , so laboratory cultures should be stored at (3-5ºc) in blood broth .
- The organism is sensitive to sulfonamides and penicillin.

- **Streptococcus toxins and enzymes:**
- It produces two soluble haemolysins which cause haemolysis of erythrocytes.
- *Strept. pyogenes* produces a leucocyte destroying substance known as leucocidine.
- Itproduces erythrogenic toxin which is responsible for the characteristic skin lesions of scarlet fever.
- Produce fibrinolysin ,proteinase, hyalouronidase (spreading factor).

- **Antigenic structure**
- Somatic (o)antigen
- Capsular antigen
- (m) protein only in s.pyogen
- **<u>Pathogenicity</u>**:
- *Strept. pyogenes* causes a number of disease in man.
- It is associated with scarlet fever, septic sore throat, tonsillitis and various suppurative inflammatory conditions.
- The organism invades the bovine mammary gland producing mastitis.
- Some strains of *Strept. pyogenes*are highly virulent for the mouse and rabbit, guinea pig is more resistant.
-

- **Laboratory diagnosis of *Strept.pyogenes* infections:**
- By direct smears of the exudates from the lesions of the disease usually reveal long chains of streptococci.
- Haemolysis of blood media.
- Lancifield technique with various biochemical tests.

- =**Differences between *Staph.aureus* and *Strept. pyogenes*:**

Characters	*Staph. Aureus*	*Strept. Pyogenes*
Arrangement	Grape like clusters	Long chains
Growth	After 24 hours	After 48 hours
Turbidity in broth	Turbid and thick sediment	Clear and granular sediment
Colonies on ordinary media	Round, convex, opaque and pigmented	Minute, transparent, non pigmented
Catalase test	+	–
Gelatin liquefaction	+	–
Methylene blue	+	–
Bactiracin	resist	Sensitive

- **MASTITIS STREPTOCOCCI:**
-
- ***Streptococcus agalactiae•***
- ***Streptococcus dysgalactiae•***
- ***Streptococcus uberis•***
- These three species of streptococcus are biochemically and serologically different; however, they are associated with bovine mastitis.

- **Morphology and staining:**

- Gram positive cocci
- Arranged in long chain(s. agalactica, s. disagalactiva) short chain (s. ubris)
- Non motile, non sporulated, non capsulated)

- **<u>Growth requirement and cultural characters:</u>**
- These organisms grow well on blood or serum agar, also the use of sodium azid crystal violet blood agar facilitates the isolation of these organisms.
- It is aerobic and microaerophilic and grow at 37 ºc.
-
- *Strept. agalactiae* and *strept. dysgalactia* usually produces flocculent growth in the bottom of the tube with clear supernatant liquid above while *Strept. ubris* produce a uniform turbidity in both medium.
- *Strept. agalactia* produces beta haemolysis on agar if it is refrigerated after 24 hours incubation at 37ºc while *Strept. dysgalactia*and *Strept. uberis*are non haemolytic

- **Biochemical reaction:**
- Indol negative, doesn't liquefy gelatin and doesn't reduce nitrate.
- *Strept. agalactiae* and *Strept. dysgalactia* do not ferment sorbitol while *Strept. ubris* ferment sorbitol.
- *Strept. agalactiae* ferment salicin while *Strept. dysgalactia* do not ferment salicin.
- Esculin hydrolysis in *Strept. ubris* is positive

- **<u>Resistance and viability:</u>**
- As *Streptococcus pyogenes*.
- **Antigenic structure and toxin:**

Somatic antigen only

Toxins

Haemolycin , leucocidin

- **<u>Pathogenicty :</u>**
- Three organismscausepranchymatousmastitis in cow, sheep and goats

- **<u>Laboratory Diagnosis:</u>**

- Isolation and identification of streptococci from milk samples.
- By **Hottis and miller** test for the detection of *Strept. Agalactiae*, this test is performed by adding 0.5 ml of sterile 0.5 % aqueous solution bromocresol purple to 9.5 ml milk. The milk is incubated 24 hours at 37ºc and the test is read where the appearance of canary-yellow colonies of bacterial growth along the walls and in the bottom of the test tube is diagnostic of *Strept. agalactiae* infection .

Differentiation between the 3species of streptoccoci caising mastitis:

Test	*St.agalactiae*	*St.dysgalactiae*	*St.ubris*
1)lancifield group	B	C	E
2)haemolysis on blood		γ(α)	γ
3) Growth on bile salt.	Well		
4)CAMP test	+		
5)Na hippurate test	+		+
4)Aesculin hydrolysis.	–		+
7) Type of mastitis.	Chronic	Acute	Subacute

STREPTOCOCCUS EQUI

- As s. pyogen

Pathogenesis

Cause strangle

	strangle	glander
	In upper respiratory tract Cause respiratory manifestation	In upper respiratory tract Cause respiratory manifestation
Age of animal affected	More than 5 years	Less than 5 years
Causitive agent	s. equi	Pseudomonus mallie

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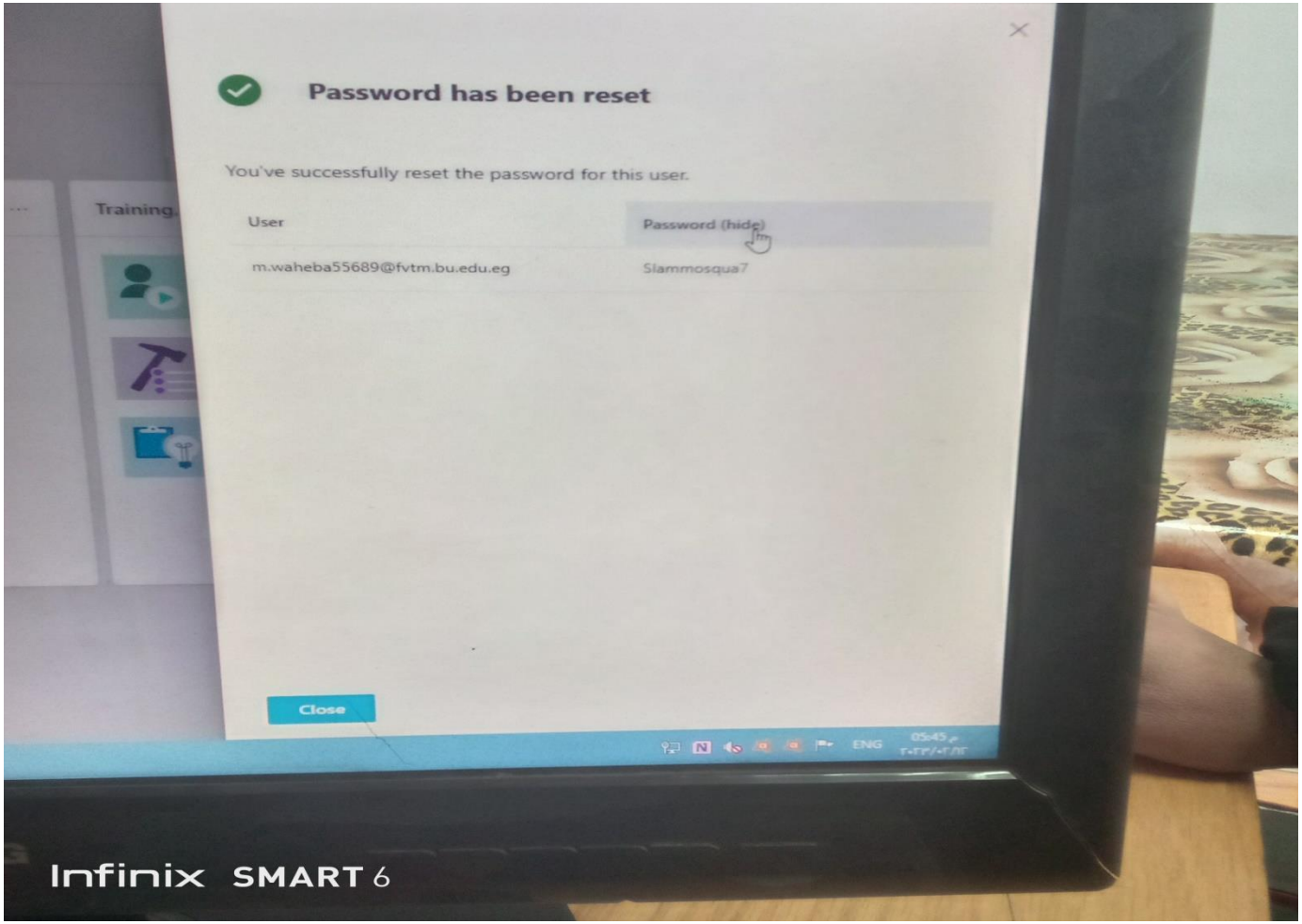
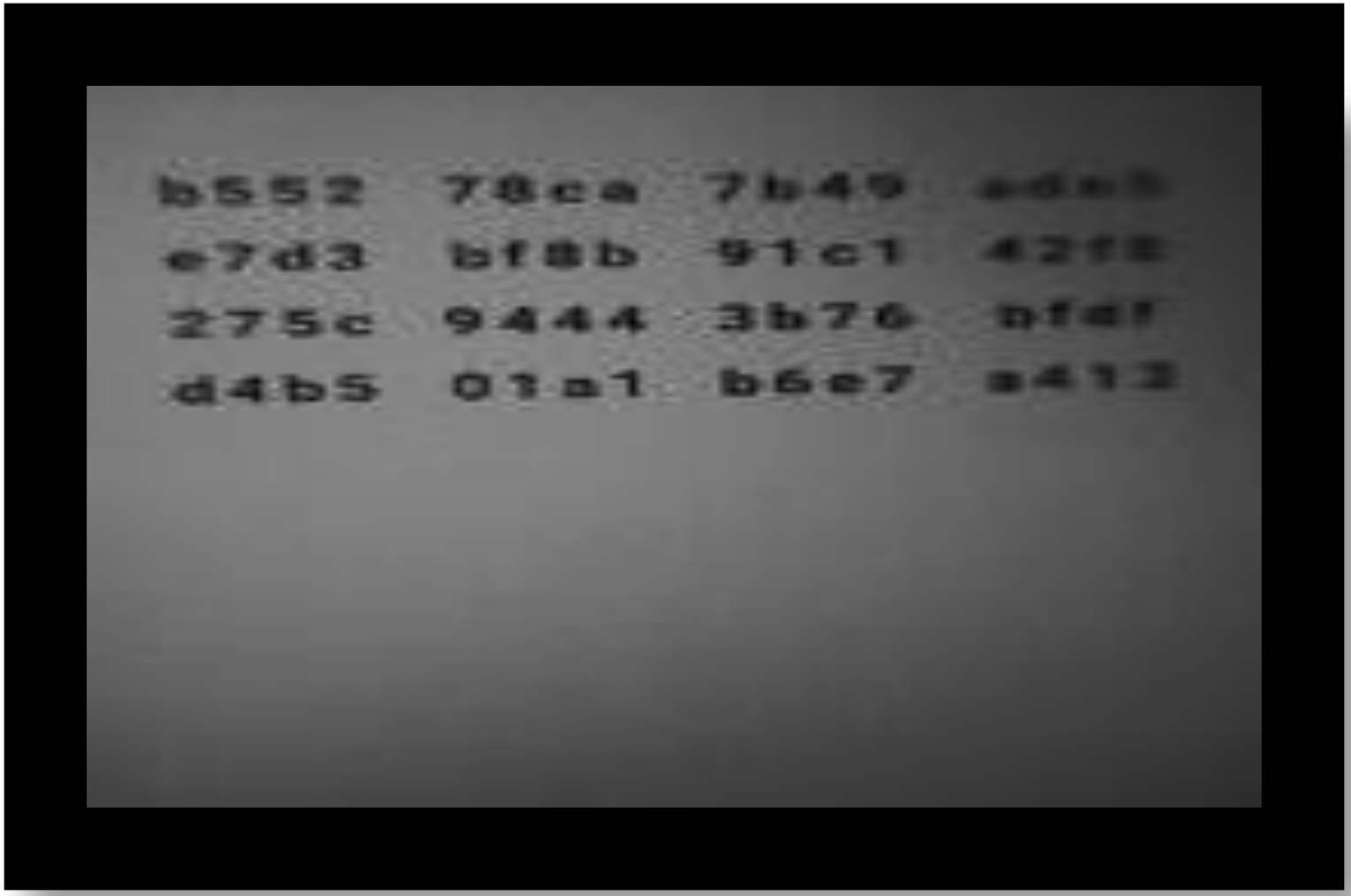

Password has been reset
You've successfully reset the password for this user.
User
Password (hide)
m.waheba55689@fvtm.bu.edu.eg
Slammosqua7
Close
Training
ENG
Infinix SMART 6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d4b5 01a1 b6e7

IMMUNOLOGY

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Bacteriology, Immunology and Mycology Dep.

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Examples of Immune Responses:

- You get the Flu;

- Vaccination;

- Atopic Allergy;

- Transplantation;

- Autoimmunity;

2 good things/3 bad things: It depends on the insult, and the timing, strength & types of responses - more good than bad - Really

Examples of Immune Responses:

Flu:

Vaccination:

Atopic Allergy:

Transplantation:

Autoimmunity:

The environment contains
avast number of infectious organsims

Viruses bacteria fungi protoza
worms

damage&
kill the host if they multiply unchecked

In fact,

they leave very little permenant damage due to:

IMMUNE SYSTEM

Which split into functional division

Innate immunity **Acquired immunity**

:

Acquired immunity

-produce a specific response

To eradicate The offending material

The adaptive immune system remembers the particular infectious agent & can prevent it causing disease later

the immune system consists of:

A number of organs

several different cell types

tissue cells

STEM CELLS IN THE BONE MARROW

W. B.CS

R . B .CS

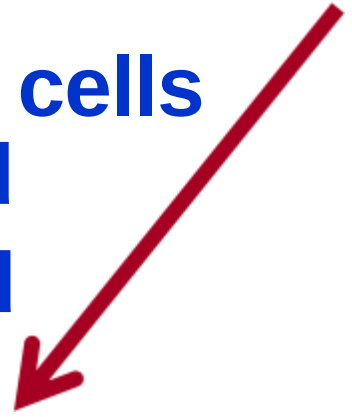
Where do the cells of the Immune System (both innate & adaptive) come from?

Hematopoiesis in the Bone Marrow:

Production of blood cells

Myeloid

Lymphoid

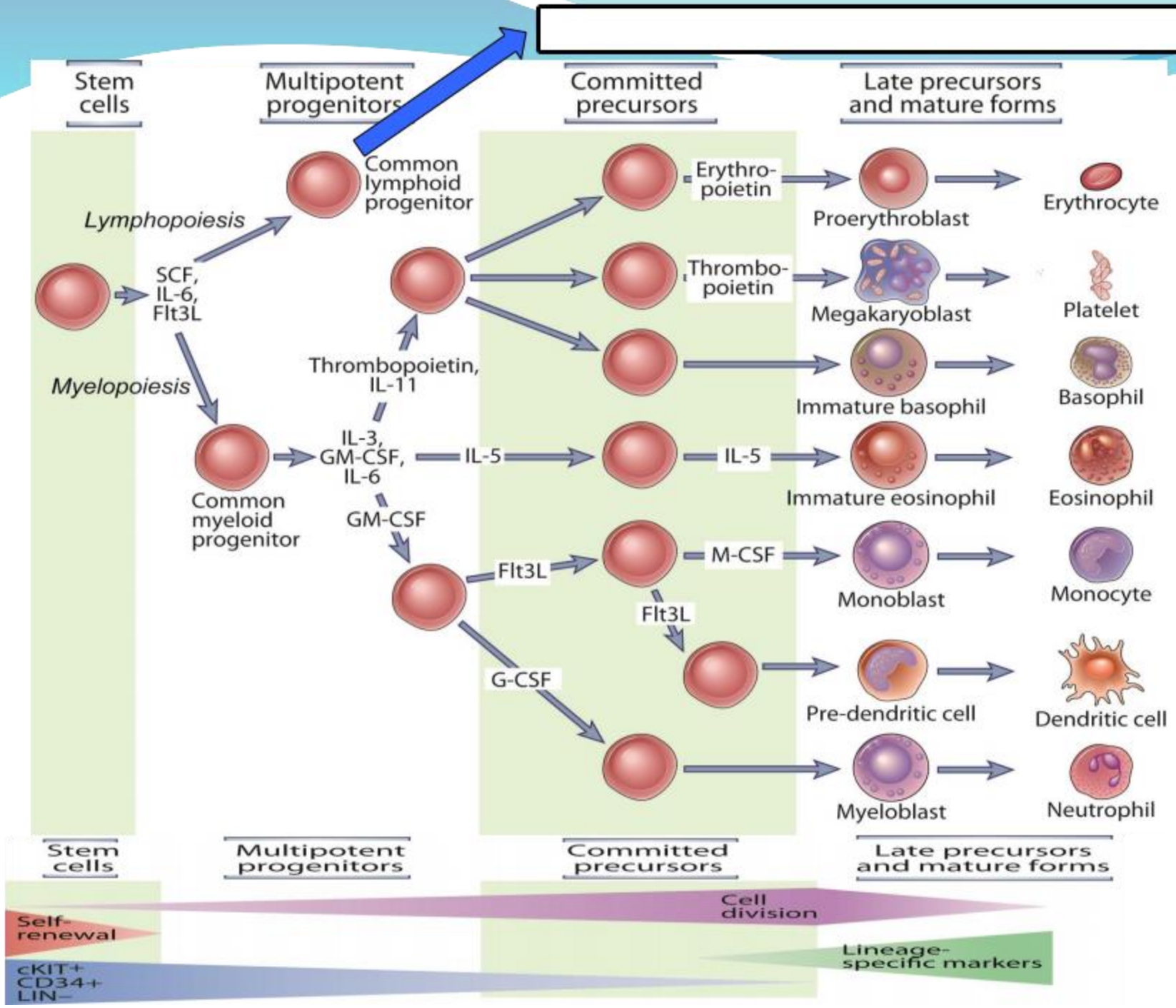


Central Lymphoid Organs

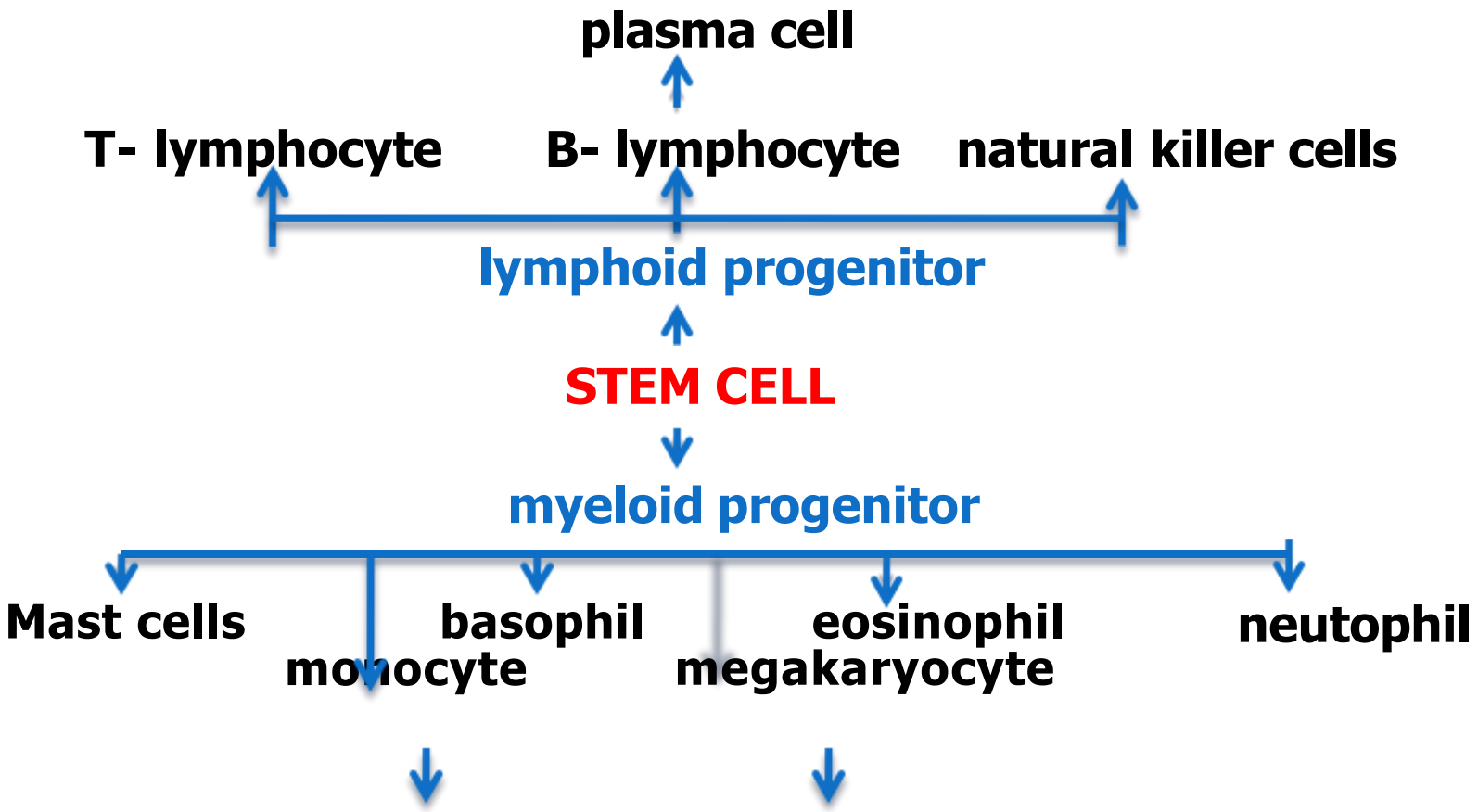
Bone marrow (Bursa of Fabricius in birds(

Thymus

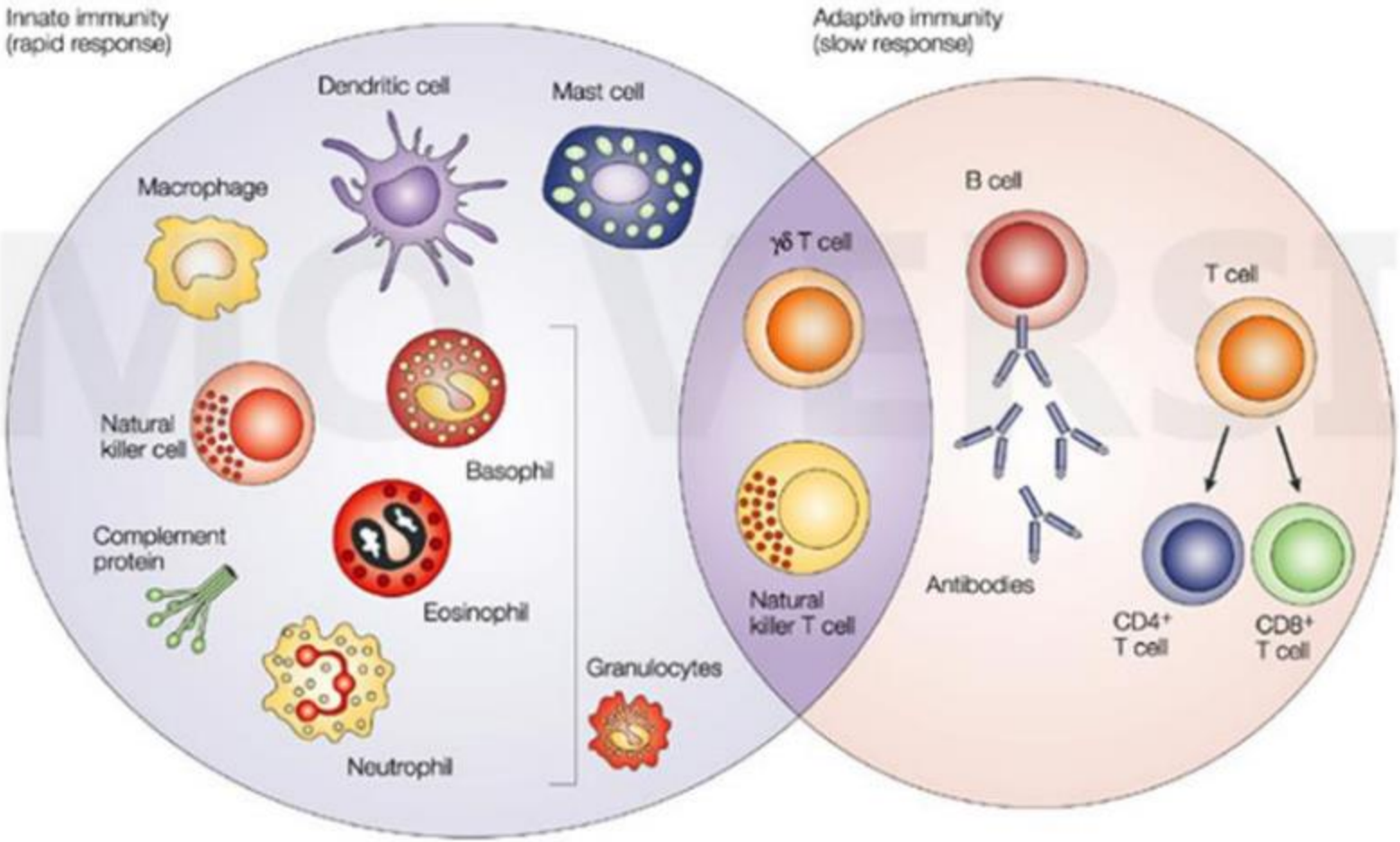
HEMATOPOIESIS



Cells of the immune system

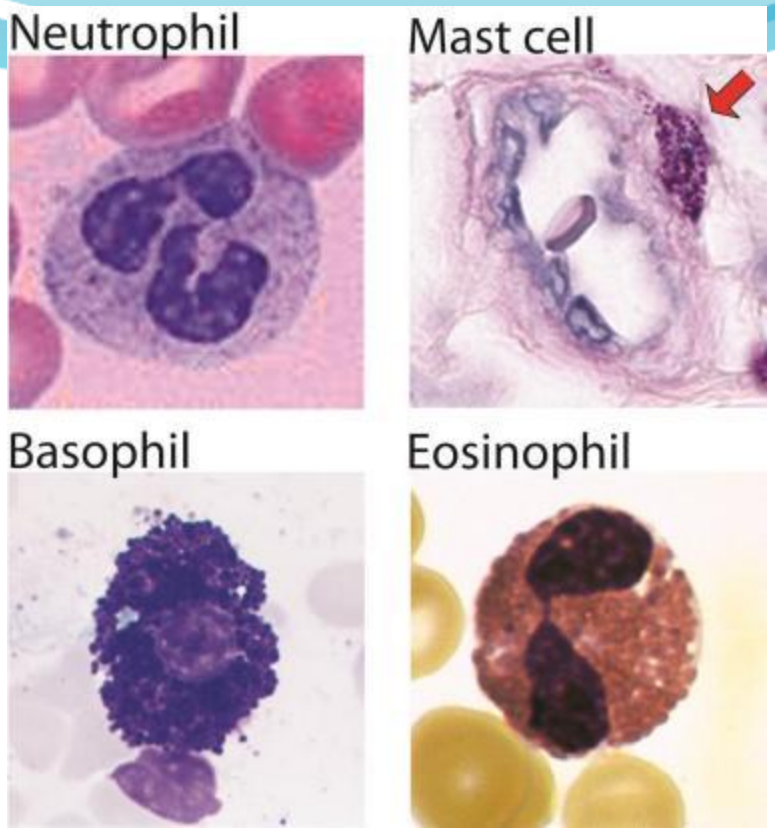


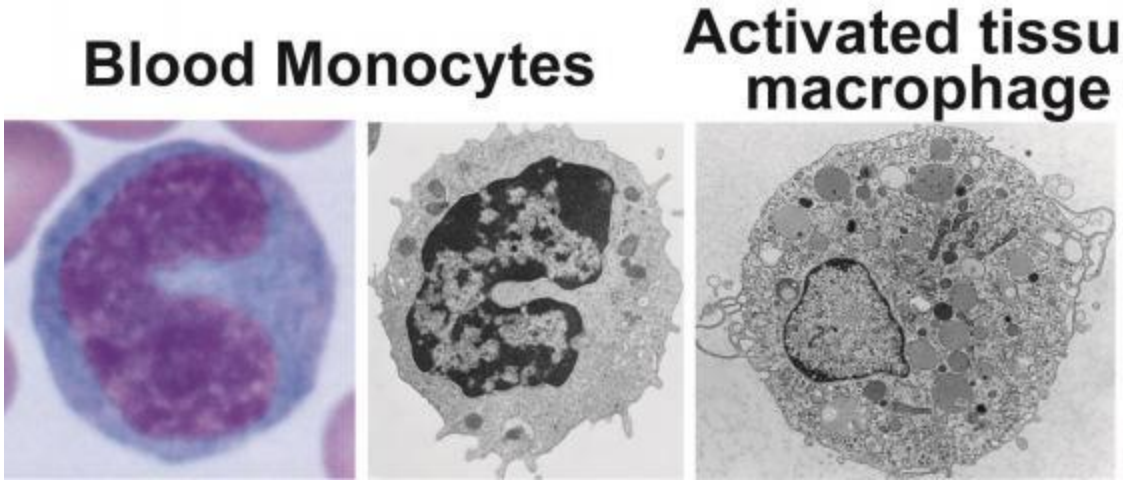
Cells of Innate and Adaptive Immunity



The non-lymphoid players:

Some participate in the induction phase and the effector phase, some only as immune response effectors





Lymphoid cells

-Lymphocytes → 20% of W .B.CS) adult(

-Mature lymphoid cells → long lived for many years

as memory cells

-The mononuclear cells are heterogenous in size& morphology

-The typical small lymphocytes are agranular& comprise → T- lymphocyte

-B- lymphocyte

-The large cells (large granular lymphocytes(
Contain cytoplasmic granules

Cells which able to kill certain tumor & virally-infected cell ⟶ natural killing & destroy cells coated with immunoglobulin

)mediated antibody - dependent cell- - cytotoxicity(

Myeloid cells

The second pathway of development

Mononuclear phagocytes polymorphonuclear leucocytes

The common myleoid progenitor → monocytes → blood → tissues → macrophages (lifespan months to years(

The human blood monocyte is larger than a lymphocyte & has a kidney shape nucleus

This actively phagocytic cell has an ruffled membrane & many cytoplasmic granules

These lysosomes contains:

Enzymes ← **molecules that are involved in the killing of M.O**

Mononuclear phagocytes adhere strongly to surface & have various cell membrane receptors to aid the binding & ingestion of foreign material

Their activities can be enhanced by molecule produced

by T-lymphocytes called lymphokines

Monocytes & macrophages

Lymphokines & monokines → cytokines

Cells of the immune system

Cell type	Developmental lineage	Morphological definition	Type of immunity
neutrophils	myeloid	Polymorphonuclear	innate
eosinophils	myeloid	polymorphonuclear	innate
basophils	myeloid	polymorphonuclear	innate
Mast cells	myeloid	polymorphonuclear	innate
Monocytes-macrophages	myeloid	mononuclear	innate
Natural killer cells	lymphoid	mononuclear	innate
Cytotoxic T-lymphocytes	lymphoid	mononuclear	adaptive
Helper T-lymphocytes	lymphoid	mononuclear	adaptive
B - lymphocytes	lymphoid	mononuclear	adaptive

Polymorphonuclear lymphocytes granulocytes

3-2days lifespan-

-divided acc. To histochemical staining- :

-1neutrophils (microphages(multilobed nucleus:

-most abundant circulating granulocytes-

-smaller than macrophages-

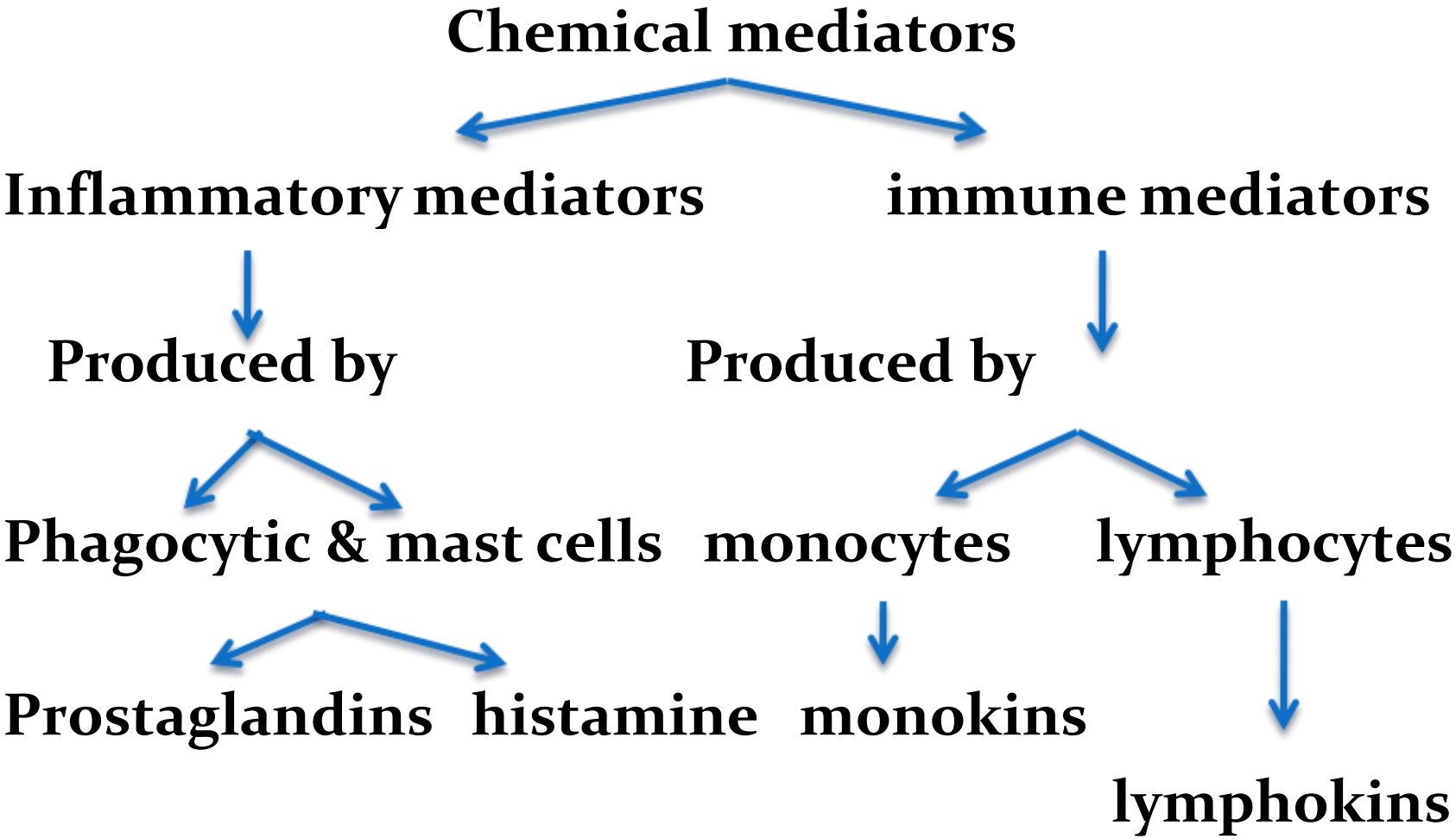
-migerate to site of infection (chemotaxis- (

i ,e the cells enter the tissues when achemotactic - produced due to infection or injury

-posses phagocytic activity-

-produce inflammatory mediators such as- prostaglandins

&immune mediators such as interleukins-


Chemical mediators
Inflammatory mediators
immune mediators
Produced by
Produced by
Phagocytic & mast cells
monocytes
lymphocytes
Prostaglandins
histamine
monokins
lymphokins

-2eosinophil (acidophil(

Less efficient than neutrophils-

%2-1 -of leucocytes-

- increase in allergic conditions -

-somewhat phagocytic-

-their major function by producing cytotoxic - molecules in granule content can kill parasites that are too large to be phagocytosed

-3basophils

Less than 0.2% of leucocytes-

-release pharmacological mediators-

Such as histamine (which responsible for allergic reaction(-

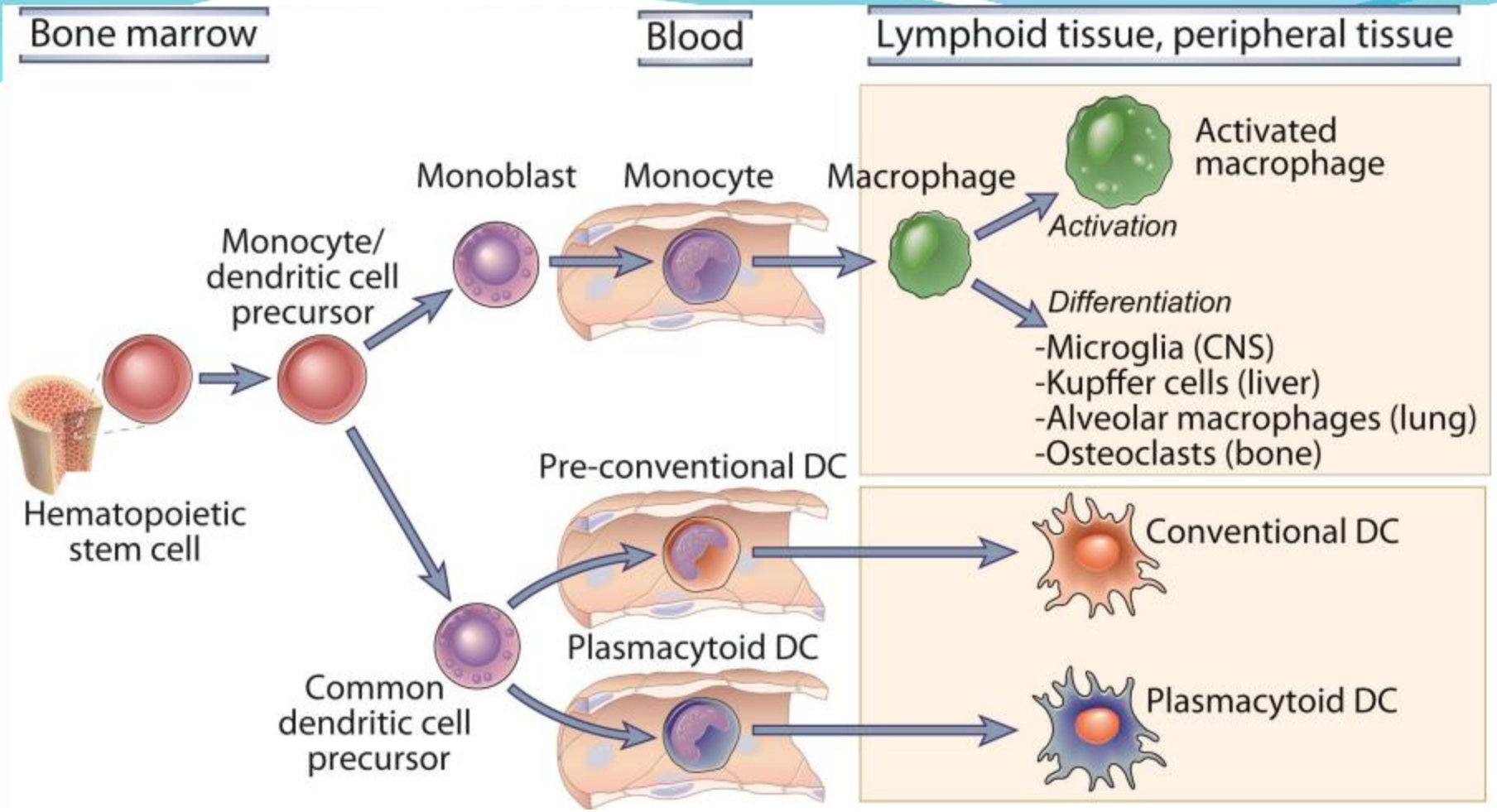
-4monocytes (macrophages(

Monocytes circulate in blood for 3 days , then migerate to tissues & change to macrophages -

-migerate to site of infection (chemotaxis- (

-posses high phagocytic activity-

-produce inflammatory & immune mediators-
-digestion of microorganism-



These mononuclear phagocytes are derived from a common precursor in the bone marrow, then differentiate to monocytes or dendritic precursors, circulate in the blood, and mature further depending on environmental cues

-5natural killer cells (NK cells(

Kill tumor cells & virus infected cells-

-non specific-

just destroy any cell that can develop tumor-

-they are called natural killer cells because the -cells kill without the need of antigen -specific activation but enhanced by exposure to certain cytokines (interferons) produced by virus infected cells

-6killer cells (K cells(

-more specific

They kill antibody - coated target cells-

-mediate antibody -dependant cellular cytotoxicity-ADCC-

MECHANISM OF KILLING- :

PERFORINS - GRANZYMES - APOPTOSIS

-7LYMPHOCYTES

There are 2 main subsets B & T lymphocytes-

B-lymphocytes	T-lymphocytes
Named after Bursa of fabricius in chickens In human, bone marrow, peyer patches & appendix	Named after thymus gland on which their maturation depends
Constitute 30% of total lymphocytes	Constitute 70% of total lymphocytes
Of short lifespan (days or weeks(	Of long lifespan (months or years) Memory cells
Upon stimulation , they differentiate to plasma cells producing specific antibody or immunoglobulins	Upon stimulation, they differentiate into effectorT-cells, some produce lymphokins, some cytotoxic & other can be helper cells

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Dermatophytes

- **Dermatophytes**

are particular group of fungi that inhabit the most superficial parts of the body including keratin of stratum corneum and hairs causing an integumentary disease called ring worm or Dermatophytosis which characterized by circular areas of alopecia, scaling, crusts.

Classification of dermatophytes:

- **Zoophilic** dermatophytes are mainly found in animals but can be transmitted to humans.
- **Anthropophilic** dermatophytes are mainly found in humans and are very seldom transmitted to animals.
- **GeophiIic** dermatophytes are found mainly in soil, where they are associated with decomposing hair, feathers, hooves and other keratin sources. They infect both humans and animals.

- There are **3 genera** of dermatophytes
  - **Epidermophyton**
  - **Microsporum**
  - **Trichophyton**.

The most important species of dermatophytes:

A- Genus trichophyton:

1- *T. mentagrophytes*: Zoophilic, it is a frequent pathogen of rodents and dogs; occasional infections of cats, horses, cattle and man are observed.

2-*T. verrucosum*: Zoophilic cause the majority of ringworm infections In cattle and occasionally involves horses, dogs and other domestic animals.

3-*T. equinum*: zoophilic, frequently infects horses, human Infections occur occasionally and canine infection occur rarely.

4-*T. gallinae*: Zoophilic, infects poultry and infrequently other birds ard man. Infection in birds is limited to the skin usually the comb and wattles and does not invade the feathers, causes favus in poultry.

B- Genus Microsporum:

1- *Microsporum canis*: zoophilic, frequently infects cats, dogs and man, horses and lower primates are infected less frequently.

2- *Microsporum nanum*: Geophilic frequently causes mild infections in swine and infrequently involves man.

Reproduction

- **On the host** it reproduces asexually by formation of arthrospores.
- **On culture media** it reproduce asexually by formation of conidia which may be small in size (microconidia) or large in size (macroconidia). That differ in size and shape between dermatophyte species.

D

I. Clinical signs:

- Scaling or crusts on affected are

- Circular areas of alopecia (hair loss), as the hairs in the affected area are brittle and break near the skin surface, often giving the lesion a “shaved” appearance; truncated hair shafts may be seen through the scales and crusts.
- the lesion is circular in shape due to its central healing and spread infection toward the periphery of the lesion so it is called “ringworm”.

II. - Preliminary examination (wood’s lamp):

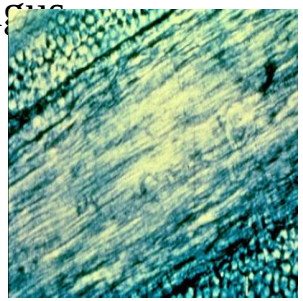
- Some dermatophytes strains, as *M. canis,* exhibit green fluorescence when stimulated by certain wavelengths of UV light.
- A Wood’s lamp can be used to examine the fur for these fungi.
- Certain topical preparations or alcohol may mask the fluorescence.

III- **Sampling**

- The samples are collected from periphery of the lesion including full thickness of keratinized epidermis until the blood is just drawn.
- The samples should be collected at paper envelope and submitted to the laboratory in inside additional warping, in dry condition.
- the site of the lesion should be washed firstly with water and detergent and allow to dry then wipe it with alcohol 70%.
- from periphery of the lesion collect skin scraping using blunt scalpel blade or microscopic slide.
- Damaged hair should be plucked from the lesion, never cut with scissors.

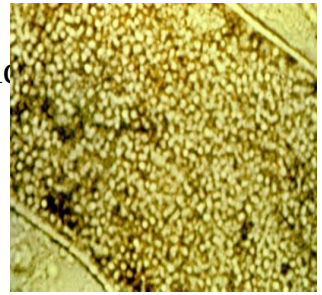
IV. Direct microscopic examination.

- On microscopic slide place the sample (hair or skin scraping) then add few drops of KOH 10-20 % to dissolve keratin layer which facilitate observation of the fungus.
- Then put cover slide and leave overnight.
- Examine under low and high power. Ectothrix
- The dermatophyte species appear as branching septate hyphae and chains of arthroconidia.



Ectothrix

- **In hair sample:**
  - Arthroconidia either accumulate outside the hair shaft) this indicate **ectothrix** arthroconidia which characteristic of all significant animal dermatophytes
  - or arranged inside hair shaft **(endothrix**) which common for anthrobophilic



Endoth

V. Culture characters:

- **Culture media used for isolation of dermatophytes:**
  - Sabouraud's dextrose agar with chloramphenicol (0.05 gm / litter) and cycloheximide (0.4 gm/litter).
  - *T. verrucosum* requires addition of thiamine and/or inositol for their growth.
  - *T. equinum* requires addition of nicotinic acid.
- Skin scrapings or plucked hair samples can be scattered over the surface of agar and gently pressed down on the medium with a swab or sterile forceps.
- Incubate aerobically at 25-30°c except *T. verrucosum* at 37°c for 3-4 weeks.

- Observe the observe and reverse colour of the colony, texture and growth rate.
- All these features are differ between dermatophyte species and it is diagnostic.

For example:

- ***T. Verrucosum*** is slow growing (3-4 weeks) and it has smooth, folded colonies. The observe side varies in colour from white or whitish grey and reverse side has white colour.

- ***T. equinum*** is Fast growing (10-14 d). Colony initially flat, white and fluffy but later velvety with central folding, cream observe colour and yellow to reddish brown reverse colou

- ***M. canis*** is fast growing (10-14 d). Its surface is white and cottony at the center with bright yellow periphery. Reverse side is bright yellow.

Microscopic examination of the colonies

- on the culture dermatophytes produce the reproductive structures (conidia) and hyphae which are characteristic for every genus and species.
- It can be observed under microscope by using a “slide culture technique” or by using adhesive side of clear cello-tape stripe and lactophenol cotton blue stain.
- The conidia may be macroconidia (multicellular) or micro-conidia (unicellular)

Slide Culture technique.

Used to observe conidia and hyphae of fungal colonies.

Procedures:

1. Using a sterile blade cut an agar block (1 x 1 cm) from a plate of Sabauroud's dextrose agar, small enough to fit under a cover-slip, and put it over a sterile glass slide.
2. Inoculate the four sides of the agar block with spores or mycelial fragments of the fungus to be grown.
3. Place a flamed cover-slip centrally upon the agar block.
4. Incubate the plate at humid chamber (put filter paper soaked with sterile dist in the peteri dish then put the slide over bended glass rod) at 26 ℃ until growth and sporulation have occurred.
5. Remove the cover slip from the agar block.
6. Apply a drop of 95% alcohol as a wetting agent.
7. Gently lower the cover-slip onto a small drop of Lactophenol cotton blue on a clean glass slide.

Differentiation between macro- and microconidia of dermatophyte genera

	Microsporum	Trichophyton	Epidermophyton
Presence	Usually present.	often absent.	present
Wall	Thick	Thin	moderately thick
Surface	Rough	Smooth	Rough
Shape (differ between species)	Ex. ***M.canis***: Spindle, mature spore end in distinct knob and of 6-15 cells.	***T.verrucosum***: Extremely rare, chains of chlamydospores are more characteristic.	***E. floccosum***: pear-shaped in clusters Septate into 2-5 cells.
Microconidia	Variable often absent if present they are tear shape and borne singly on hyphae.	Usually numerous and borne singly along hyphae or in grape like clusters.	absent

Order: EUBACTERIALIS

Family: LACTOBACTERIACE

Genus: STEROTOCOCCUS

SPECIES:

Sterptococcus pyogenes

Sterptococcus agalactiae

Sterptococcus dysagalactiae

Sterptococcus uberis

Sterptococcus equi

Sterptococcus zooepidemicus

Sterptococcus canis

Sterptococcus gallinerum

General characters of Streptococci:

- It is spherical or ovoid cocci in shape 0.5 µ in diameter, it is usually arranged in chains of various length, gram positive .they are non motile, non sporulated but some strains are capsulated (slime layer).

- **<u>Growth requirements and cultural characters:</u>**
- Streptococci are aerobic or facultative anaerobic, grow weak on ordinary media but grow well on blood or serum agar at 37ºc and ph 7,2 . The organism produces haemolysis on blood agar plate the specific media are ***Edward´s blood ager*** media and sodium azide agar media.
- Streptococcus doesn't produce pigments either in fluid media or on solid media. The colonies on blood agar are small, smooth – glistening after 48 hours, while in broth granular growth and powdery sediment are formed within 36-48 hours.

- **<u>Classification:</u>**
- **1-according to type of haemolysis :**
- Streptococci causing complete haemolysis (Beta (β) haemolysis), a clear zone of haemolysis as *Strept.pyogenes* and *Strept.equi*.
- Streptococci causing in complete haemolysis (alpha α) such as *Strept.viridians* and *pneumococci*.
- C) Streptococci causing no haemolysis (gamma γ), non hemolytic streptococci as *Strept.lactis* and *Sterpt.salivaris*.

- **2-Lancifield classification:**
- Lancefield reported that the hemolytic streptococci could be divided into groups (A-N) on serological basis depending on precipitation test using the specific carbohydrate substance designated as C.substance as the antigen which differed in each group:
- - Group A: *Streptococcus pyogenes*
- - Group B: *Streptococcus agalactia*
- -Group C: animal streptococci as *streptococcus zooepidemicus*, *Streptococcus dysgalactia, Streptococcus equi, Streptococcus canis.*
- -Group D: Enterococci as streptococcus fecalis ,*Streptococcus faecium*
- -Group E: Strains isolated from milk and dairy products as *Streptococcus uberis*
- -Group F: Strains isolated from respiratory tract.
- -Group G: Strain isolated from respiratory tract of man and dogs.
- -Group H, K, M and group N

- **3-Sharman classification:**
- It depends on the biochemical characters of the enterococci or non pathogenic streptococci on the following reactions:
- Growth at 10 ºc and 45ºc as *Strept.viridians*.
- Tolerance to 6.5 % sodium chloride.
- Reduction of methylene blue.

- ***Streptococcus pyogenes***
- **Morophological characters:**
- *Strept.pyogenes* are arranged in long chain, it is cocci , non motile , non sporulated, has capsule in young culture and gram positive
- **Distribution:**
- It is widely distributed on the skin and in the mucous membrane

- **Growth requirements and cultural characters**:
- *Strept.pyogenes* grow on media containing blood or serum at 37ºc and PH 7.2 .
- Its colonies on the solid media are very small, smooth glistening, dew drop like, finally granular and not pigmented.
- The colonies produce zone of complete haemolysis (beta haemolysis)on blood agar after (24-48) incubation

- **Biochemical properties:**
- The organism ferments glucose, lactose,sucrose, and salicin producing acid only do not ferment sorbitol, do not liquefy gelatin, catalase negative and indol negative.
- **Resistance:**
- *Strept. pyogenes* killed by 60ºc for half hour , so laboratory cultures should be stored at (3-5ºc) in blood broth .
- The organism is sensitive to sulfonamides and penicillin.

- **Streptococcus toxins and enzymes:**
- It produces two soluble haemolysins which cause haemolysis of erythrocytes.
- *Strept. pyogenes* produces a leucocyte destroying substance known as leucocidine.
- Itproduces erythrogenic toxin which is responsible for the characteristic skin lesions of scarlet fever.
- Produce fibrinolysin ,proteinase, hyalouronidase (spreading factor).

- **Antigenic structure**
- Somatic (o)antigen
- Capsular antigen
- (m) protein only in s.pyogen
- **<u>Pathogenicity</u>**:
- *Strept. pyogenes* causes a number of disease in man.
- It is associated with scarlet fever, septic sore throat, tonsillitis and various suppurative inflammatory conditions.
- The organism invades the bovine mammary gland producing mastitis.
- Some strains of *Strept. pyogenes*are highly virulent for the mouse and rabbit, guinea pig is more resistant.
-

- **Laboratory diagnosis of *Strept.pyogenes* infections:**
- By direct smears of the exudates from the lesions of the disease usually reveal long chains of streptococci.
- Haemolysis of blood media.
- Lancifield technique with various biochemical tests.

- =**Differences between *Staph.aureus* and *Strept. pyogenes*:**

Characters	*Staph. Aureus*	*Strept. Pyogenes*
Arrangement	Grape like clusters	Long chains
Growth	After 24 hours	After 48 hours
Turbidity in broth	Turbid and thick sediment	Clear and granular sediment
Colonies on ordinary media	Round, convex, opaque and pigmented	Minute, transparent, non pigmented
Catalase test	+	–
Gelatin liquefaction	+	–
Methylene blue	+	–
Bactiracin	resist	Sensitive

- **MASTITIS STREPTOCOCCI:**
-
- ***Streptococcus agalactiae•***
- ***Streptococcus dysgalactiae•***
- ***Streptococcus uberis•***
- These three species of streptococcus are biochemically and serologically different; however, they are associated with bovine mastitis.

- **Morphology and staining:**

- Gram positive cocci
- Arranged in long chain(s. agalactica, s. disagalactiva) short chain (s. ubris)
- Non motile, non sporulated, non capsulated)

- **<u>Growth requirement and cultural characters:</u>**
- These organisms grow well on blood or serum agar, also the use of sodium azid crystal violet blood agar facilitates the isolation of these organisms.
- It is aerobic and microaerophilic and grow at 37 ºc.
-
- *Strept. agalactiae* and *strept. dysgalactia* usually produces flocculent growth in the bottom of the tube with clear supernatant liquid above while *Strept. ubris* produce a uniform turbidity in both medium.
- *Strept. agalactia* produces beta haemolysis on agar if it is refrigerated after 24 hours incubation at 37ºc while *Strept. dysgalactia*and *Strept. uberis*are non haemolytic

- **Biochemical reaction:**
- Indol negative, doesn't liquefy gelatin and doesn't reduce nitrate.
- *Strept. agalactiae* and *Strept. dysgalactia* do not ferment sorbitol while *Strept. ubris* ferment sorbitol.
- *Strept. agalactiae* ferment salicin while *Strept. dysgalactia* do not ferment salicin.
- Esculin hydrolysis in *Strept. ubris* is positive

- **<u>Resistance and viability:</u>**
- As *Streptococcus pyogenes*.
- **Antigenic structure and toxin:**

Somatic antigen only

Toxins

Haemolycin , leucocidin

- **<u>Pathogenicty :</u>**
- Three organismscausepranchymatousmastitis in cow, sheep and goats

- **<u>Laboratory Diagnosis:</u>**

- Isolation and identification of streptococci from milk samples.
- By **Hottis and miller** test for the detection of *Strept. Agalactiae*, this test is performed by adding 0.5 ml of sterile 0.5 % aqueous solution bromocresol purple to 9.5 ml milk. The milk is incubated 24 hours at 37ºc and the test is read where the appearance of canary-yellow colonies of bacterial growth along the walls and in the bottom of the test tube is diagnostic of *Strept. agalactiae* infection .

Differentiation between the 3species of streptoccoci caising mastitis:

Test	*St.agalactiae*	*St.dysgalactiae*	*St.ubris*
1)lancifield group	B	C	E
2)haemolysis on blood		γ(α)	γ
3) Growth on bile salt.	Well		
4)CAMP test	+		
5)Na hippurate test	+		+
4)Aesculin hydrolysis.	–		+
7) Type of mastitis.	Chronic	Acute	Subacute

STREPTOCOCCUS EQUI

- As s. pyogen

Pathogenesis

Cause strangle

	strangle	glander
	In upper respiratory tract Cause respiratory manifestation	In upper respiratory tract Cause respiratory manifestation
Age of animal affected	More than 5 years	Less than 5 years
Causitive agent	s. equi	Pseudomonus mallie

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DEPARTMENT OF ZOONOSES

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مجموعة ١٣١٣

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جمهورية مصر العربية
بطاقة تحقيق الشخصية
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محمود محمد يوسف وهيبه
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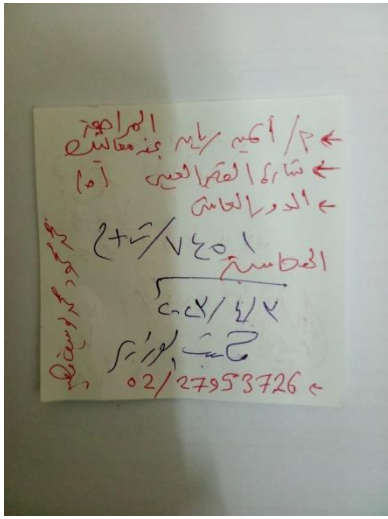
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EPIDEMYOLOGY OF BRUCELLA

DEMANDS OF LOBNA SALEM

DEPARTMENT HEAD

ZOONOSIS

BACTERIOSIS

BRUCELLOSIS

BRUCELLOSIS

1-BRUCELLOSIS IS AZOONOSIS

AWORLD WIDE DISTRIBUTION

2-ACUTE AND OR CHRONIC DISEASE

3-MAY CAUSE APHYSICAL IN CAPACITY

4- HAVE MANY SYNONYMS

4- HAVE MANY SYNONYMS INCLUDES :

A-SYNONYMS OF INFECTIONS IN HUMANITY :

-1-UNDULENT FEVER 11- MALTA FEVER 111- MEDITERRANEAN FEVER

B-SYNONYMS OF INFECTIONS IN ANIMALS :

1- CONTAGIUOS ABORTIONS IN ANIMALS

11-BANG S ABORTIONS IN CATTLE

5-CAUSTIVE ORGNISMS :

THREE MAJOR SPECIES OF BRUCELLAE INCLUDES :

1-BRUCELLA ABORTUS

2- BRUCELLA MELITENSIS

3-BRUCELLA SUIS

4-RECENTLY BRUCELLA CANIS

BRUCELLA ABORTUS OF CATTLE: CUSING ABORTION OF CATTLE AT 6-10 MONTH OF PREGENANCY

BRUCELLA MELITENSIS OF GOATS AND SHEEP : CAUSING ABORTIONS IN THESE ANIMALS AT 3 TO 4 MONTHS OF PREGENANCY BUT MORE SEVER INFECTIONS IN HUMANS AND MAY BE FATAL ESPECIALLY IN YOUNG AND ELDERLY AGES BUT ABOTION IS LESS COMMON ON ALARGE SCALES IN ANIMALS AS ACOMPARISONS OF RUMINANTS

BRUCELLA SUIS OF PIGS : CAUSING ABORTION OF DOGS ABOUT 50 DAYS OF GESTATION

BRUCELLA CANIS : GIVES COMPLETE HOMOLOGY WITH BRUCELLA SUIS

BRUCELLA CANIS RARELY INFECT MAN BUT SLIGHTLY INVASIVE FOR HIM

5-BRUCELLAE ARE RELATIVELY RESISTANCE IN THE ENVIRONMENT
6-BUT READILY DESTORYED BY PASTEURIZATIONS AND BOILING OF MILK AND CHLORINATION OF WATER
7-PATHOGENICITY :
N.B-THE 4 BRUCELLA SPECIES ARE PATHOGENIC TO MAN BUT THEIR VIRULENCE VARIES IN THE ORDER OF MELITENSIS ,SUIS , ABORTUS , CANIS.
8-RESERVOIRS OF INFECTIONS :
1-BRUCELLA IS AZOONOSES PAR EXELLENCE WHY :-
A-THE ROLES OF ANIMALS ARE ESSENTIALS IN TH E PERSISTANCES OF INFECTIONS .
B-HUMAN TO HUMAN TRANSMISSION ARE EXCPTIONAL .
C-THE ORIGINAL RESERVOIRS INCLUDE :
A-GOATS B- SHEEP C-PIGS D- DOGS
D- THE MINOR RESERVOIRS INCLUDES :
A-HORSES B- FOWEL (ARE YOU SURE) C- SOME SPECIES OF RODENTS
MAJOR AND MINOR RESERVOIRES ARE CONSIDERED AS ASOURCES OF HUMAN INFECTIONS

9- ORIGINAL RESERVIORS CAN BE INFECTED WITH OTHER SPECIES OF BRUCELLAE BESIDE THE SPECIFIC SPECIES :

E.G A- CATTLE CAN ALSO BE INFECTED BY BRUCELLA SUIS AND BRUCELLA MELITENSIS

B-GOATS AND SHEEPS CAN BE INFECTED BY BRUCELLA SUIS AND BRUCELLA ABORTUS

C-SWINE CAN BE INFECTED BY BRUCELL ABORTUS

D-DOGS CAN BE INFECTED BY BRUCELLA ABORTUS AND BRUCELLA SUIS AND BRUCELLA MELITENSIS

10-BRUCELLOSIS IN HORSES USUALLY MANIFESTS ITSELF IN THE FORM OF :

1- FISTULOUS BURSITIS (POLL EVIL) AND FISTULOUS WITHERS .

2-ABORTION ARE IN RARE IN HORSES .

3-MAN CAN CONTRACT THE INFECTIONS FROM HORSES WITH OPEN LESIONS .

11-EPIDEMYLOGICAL STUDIES OF BRUCELLOSIS DURING THE LAST DECADES PROVED THAT NOT ONLY DOMESTIC ANIMALS ARE THE MAIN CARRIERS BUT ALSO 24 SPECIES OF WILD ANIMALS AND 18 SPECIES OF BLOOD SUCKING ARTHROPODES CAN HARBOUR THE BRUCELLAE AND CATS ARE RESISTANCE TO BRUCELLA AN NO CASES OF NATURAL DISEASE OCCUERENCE ARE KNOWN

12- ORGANISMS ARE FOUND IN : EXCRETA ,DISCHARGES,TISSUES

TISSUES INCLUDING : MUSCLES ,MAMMARY GLANDS , FOETUS ,PLACENTA AND VAGINAL DISCHARGES OF ABORTED ANIMALS

13- SOURCE OF INFECTIONS AND MODE OF TRANSMISSION:

MAN IS INFECTED BY ANIMALS THROUGH :

1-DIRECT CONTACT

2-INDIRECT CONTACT INCLUDES (FOOD BORNE INFECTION AND INHALATION OF AIR BORNE AGENTS)

1-FOOD BORNE INFECTION INCLUDE :

A-INGESTION OF RAW OR INSUFFICIENTLY HEATED MILK AND MILK PRODUCTS MADE OF IT ESPECIALLY FRESH CHEESE ,ICE CREAM , CREAM AND YOUGHERT NOT COMPLETELY FERMENTED B-THE ORGANISMS RARELY SURVIVE IN SOUR MILK ,SOUR CREAM ,BUTTER AND FERMENTED CHEESES AGED BY OVER THREE MONTHS .

b-the relatively scarcity of cases among children inspite of large consumption of raw milk suggestes that certain antarceologic factors of resistance are present as (lymphoid tissues more active in childerns and gastric acidity)

c-but in adults brucella was not correlated with achlorohydria , it is presumed that the organisms pass through the mucous membranes of oropharaynx,not through the stomach (are you sure) .

d-ingestion of insufficiently cooked or processed meat

e-brucellae are resistance to pickling and smoke curing thus giving rise to the possibility that some meat products so inefficient prepared could cause human infections how ever this mode of transmission has never been verified

d-it is also possible for raw vegetables and water contaminated with the excreta of infected animals to serve as sources of infections.

2-contact infection :

a-the organisms find the entry through the skin and the mucous membranes with abrasions or intake

b-brucellae produce proteolytic enzymes which ease infections through intaked skin and mucous membranes

c-brucellosis is ,for the most part ,an occupational disease of stock yard and slaghter house workers ,,butchers , veterinarians

d-the infection is usually contracted by handling fetuses and after birth ,or by contact with vaginal secretions ,excreta, and carcasses of infected animals .

e-contact infections from horses with open lesions of fistulous bursitis must be not ignored

f-contact infections in laboratories is possibles

g-several veterinarians accidentally inoculated during vaccinations of heifers with strain -19 of brucella abortus (living attenuated vaccine) as for example ,when using a defective syringe ,accidentally sprayed some vaccine in the eye as a result those veterinarians had contact the infection and developed acute brucellosis .

h-contact infections constitutes about from 60-70% of cases among veterinarians , butchers , farmers .

3-air borne infection include :
a-in and around the infected farms on the inhalation of dust of dried animals tissues and excreta
b-the minimum infective dose for humans by the way of the respiratory passages seems to be small.

4-man to man transmission :

It is very rare ,however ,a case was reported after blood transfusion ,and another case in achild from his mother .

Susceptibility : includes :

1-age 2-sex 3-occupation 4-season

1-age :

a-most frequently affected are in between 6-40 years

2-sex:

Males are infected more frequently than females (ratio 3:1) ,simply because dairy workers and meat handlers in slaughtered houses are mostly males .

3-occupation :

The disease is commonly confined to veterinarians ,farm workers or farmers (handling milk products as well as diseased animals) ,slaughter houses workers and laboratory workers .

4-season:

The disease is most prevalent in summer .these is an elevation of cases in april and reaching it is maximum in july and august

Sequelae of infection :

a-in apparent (latent infection)infection b- manifest disease

incubation period :

varies ,usually from 5-21 days ,but some times ,may be several months .it is difficult to ascertain ,onest is insidious and date of infection usually not established .

the disease in man :

a-the disease is a septicemic with sudden or insidious onest
b-is accompanied by continued , intermittened or irregular fever
c-the fever is prolonged with frequent remissions in which fever persists for 10-14 days followed by to 2-3 weeks a febrile period d-the symptomatology of the acute brucellosis ,like that of any other febrile diseases ,includes chills ,profuse sweeting and elevation of temperature .
f-an almost constant symptoms is weakness
g-any exercise produces pronounced fatigue
h-the temperature can vary from normal in the morning to 40 celicius degree in the afternoon .
i-sweeting is characterized by apeculair odour occurs at night

j-common symptoms are insomania ,sexual impotence , constipation,anorexia ,headache ,muscular and articular pains ,and general malaise .
k-this disease has amarked effect on the nervous system evidenced by by irritation ,nervousness and depressions.
l- many patients have enlarged peripheral lymph nodes or splenomegaly and often hepatomegaly ,but rarely jaundice.
m-brucella organisms localizes intracellulary in tissues of reticuloendothelial system,such as lymph nodes ,bone marrow ,spleen and liver .
n-tissues reactions are granulomatous .
o-the duration of disease can vary from aseveral few weeks or months to several years.

What are the serious complications of humans brucellosis $$$!!!!!

1-encephalitis 2- meningitis 3- peripheral neuritis 4-sponylitis 5-suppurative arthritis 6-endocarditis .

(my first clinical practical diseased case alone in a farm)

p-the chronic form of the disease occurs in some patients and may last for many years with or without the prescence of localized foci of infections . the symptoms are associated with hypersensitivity .

q-few cases of abortion in women and orchitis in man have been reported

r-persistence of infection associated either with acute relapses or chronic disease .

HISTORY OF BRUCELLA :

1-brucella melitensis discovered in island of malta in 1887 by bruce ,where a lot of soldiers died when they have drunk milk from goats ((malta fever)).

2-when the soldiers were forbidden to drink raw milk ,the disease disappeared .

3-the disease has wide distribution especially in Mediterranean countries ((Mediterranean fever)) .

4-the first case of undulant fever were reported in Egypt in by the ministry of public health in 1929.

DIAGNOSIS :

1-in man aclinical diagnosis of brucellosis based on symptoms and history should always be confirmed in the laboratory .
2-isolation and typing of casual agent is definitive and more over may indicate the source of infection
3-blood taken while the Patients is febrile is cultured on the appropriated media
4-sampling can also be taken from:
a-lymph nodes b-cerebrospinal fluids c-abscesses
5-it is recommended that the cultures be repeated several times especially in enzootic areas of brucella abortus .
6-due to the wide spread of usage of antibiotics before diagnosis in febrile patients ,bacteriologic examination often yield negative results and serologic tests become increasingly necessary.
Spread of disease in man in endemic areas :
1-sporadic cases and occasionally outbreaks (are you insists)
2-brucellin test is an intra dermaly hypersensitivity test which can be applied in survey studies to show survey studies to show prevalence of infection in man :
a-positive reaction means : past infections.
b-the intradermal test with non cellular allergens is useful for epidemiologic studies but not for clinical diagnosis.

Prevention and control :

The most rational approach for preventing human brucellosis is the control and eradication of the infection in animals reservoirs.

Measures for animals :

1-to prevent spread of infection by :
a-sanitary clean animal environment .
b-veterinary care
c-management of aborted animals and sanitary disposal of wastes.
d-in regions or countries with alow prevelance of bovine brucellosis (up to 15 %) what the excellents works :
a-an eradication program can be carried out by repeated serologic diagnostic tests applied to the entire herd
b- elimination of reactors until all foci of infection have been disappeared .
c- this procedures can be used alone in countries with alow prevalence or in combinations with the vaccinations of calves if you agood intelligences scanners.
d-epidemiologic surveillance and control and control of animal transport are very important in a such program .
e-vaccination is recommended for control of bovine brucellosis in enzootic areas with high prevalence rates (over 15 %),the vaccine of choice is brucella strain 19 confirmed by its world wide use ,the protection it gives for the useful life time of the animals and of low cost.

What are the advantages of vaccinations of brucella abortus strain 19 :

1-world wide use. 2-low cost.3-the protections it gives for the useful lifetime of the animals.

4-to avoid interference with the diagnoses it is recommended that the vaccinations be limited to young animals their ages between (calves of 3-4 months) .(why)!!!!because those animals rapidly lose the antibodies produced in response to the vaccines (memory cells eternal) .

5-it is estimated that 65% to 85% of vaccinated animals derive long term protection against infections.

6-the anti abortive effect of the vaccine is pronounced ,thus reducing one of the principle sources of the infections .

7-male calves and females over 8 months of age should not be vaccinated .

8-where possible , this upper limit should be 6 months.

9-revaccination is not recommended .

10-the objectives of a systematic and compulsory program of vaccination of calves in a given area or countery is to reduce the infection rate and obtain herds resistance to brucellosis.

11-so that , eradication of the disease may then begin .

12-it is estimated that 7-10 years of systematic vaccination are necessary to achieve this objective.

13-untill a few years ago the vaccination of adult cows with strain 19 was in advisable ,because of the prolonged persistence of antibodies that could interfere with diagnosis ,

14-several researchers proved that vaccination of adult animals with a smaller could impart an immunity comparable to that of a full dose ,while at same time agglutination titers stayed lower and disappeared faster.
15-complementary tests were very useful in distinguishing between reactions due to infection and those due to vaccinations.(by repeated serologic tests ,if the titer is increases it is an infection and if not or become lower it is vaccination (are you sure).
16-vaccinations of adult females may be considered in herds suffering acute brucellosis characterized by abortions and rapidely spreading infections and also as well as in large herds where chronic brucellosis has proven hard to eradicate .
17-the recommended dose is one to three billions cells of strain 19 brucella administered subcutaneously .
18-only animals testing should be vaccinated and they should be indelibly marked under government supervision.
19-at the beginning of the operation ,reactors should be eliminated immediately .
20-vaccinated animals should be examined serological 6 months later using rose Bengal ,mercapto ethanol and standard tube agglutination test .
21-these tests used also in diagnosing infections .
22-rose Bengal is screening test then standered tube agglutination test followed by mercapto ethanol test and those that have become infected should be send to slaughter .
23-using periodic serologic examination ,it is estimated that a problem herd can be free from infection in two years.
23-oral vaccination with strain 19 brucella is anew possibility (are sure from molecular biosynthesis) .

24-control of the infection caused by b.melitensis in goats and sheep is based mainly on vaccinations .
25-the preferred vaccine is brucella melitensis rev – 1 ,which is administered to 3-6 month old females.
26-the adult females animals can receive asmaller dose of the same vaccine (20,000 times fewer bacterial cells than the dose for young females).
27-how ever the control procedures of diagnosis and sacrificing reactor animals has produced satisfactory results in areas of low prevalence .
28-the control of swine brucellosis ,depends on test and slaughter policy as amean of eradications , no vaccines available for swines .
29-brucellosis caused by brucella canis in dogs kennels can be controlled by repeated serologic tests and blood cultures ,followed by eleminations of reactor animals .no vaccines are yet available .stray dogs must be eliminating.

Measures for man :

1-prevention of ingestion infections 2-prevention of occupational contact infections 3-prevention of air borne infections

Treatment :

1-treatment of infected persons by using :

a-tetracycline ,streptomycin , vitamin complex ,high protein diets , bed rest ,for at least three weeks .

b-cortisone in the first three days of treatment.

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

UUWD89J308ULRP4

الشكوى الإدارية فى ملفات الرقابة الإدارية المصرية

BENHA UNIVERSITY

FACULTY OF VETERINARY MEDICINE

DEPARTMENT OF BACTERIOLOGY, IMMUNOLOGY, AND MYCOLOGY

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Ministry Of Agriculture And Land Reclamation

Arab Republic Of Egypt

ترقى ماجيستير.pdf

BACTERIA sSECTION.pdf

What is the difference between PhD and Doctor of Philosophy؟

The PhD, also known as the Doctor of Philosophy ، is a research degree, which is one of the most common types of doctoral degrees ، and is awarded to graduates in many different fields

What is a PhD in philosophy called

A Doctor of Philosophy (PhD, Ph.D., or DPhil; Latin: philosophiae doctor or doctor philosophiae) is the most common degree at the highest academic level awarded following a course of study.

How long is a PhD in philosophy?

five year

The Doctor of Philosophy (PhD) is a five year program that will prepare you for an academic career in research and teaching in philosophy.

Is a PhD in philosophy difficult? Earning a PhD in philosophy takes hard work and perseverance. The process of getting your PhD in philosophy varies depending on the university and program. However, almost all programs will require coursework, intense research, and a completed dissertation. الرسالة

Is a doctorate better than a PhD?
Is a PhD Higher Than a Professional Doctorate? No, a PhD is not a higher degree than a professional doctorate. If you're wondering what's better than a PhD, both PhDs and professional doctorates are the highest level of college education that can be earned. Both degrees are doctoral degrees.

How do you address a Doctor of Philosophy?

D. When you are addressing a person with a doctoral degree, it is considered more polite to use the title Dr. or the academic abbreviation PhD with the person's name, instead of the simple courtesy titles Mr. or Ms.

What job does a Doctor of Philosophy do?

Professionals with a doctorate in philosophy often begin careers as professors at colleges and universities. Philosophy doctoral degrees may also lead to administrative roles in higher education or leadership positions in business or government settings.

What do doctors of Philosophy do?

What is a Ph. D.? A Ph. D., or Doctor of Philosophy, is a high-level degree earned after a period of three or more years of graduate-level study, culminating in the creation, submission, presentation and defense of a research dissertation.

بلغت ذروتها –إستسلام -الرسالة

Do Philosophy majors make any money?

While the starting salary is near the middle of all majors, the average mid-career salary of philosophy majors quickly rises to $81,200.00, outperforming Finance, International Relations, Marketing, Business Management, Communications, Nursing, Biology, Health Care Administration, and many others.

التخصصات-

Why is it called Doctor of Philosophy? Studies in what once was called philosophy are now classified as sciences and humanities. Hence, most disciplines, other than few like medicine and law, have their roots in Philosophy. Accordingly the name of degree awarded is Doctor of Philosophy.Jun 30, 2020

التخصصات

Is there a demand for philosophy professors?

Philosophy professor salary and job outlook The BLS predicts employment opportunities for philosophy and religious studies postsecondary teachers may increase by 7% from 2019 to 2029.Aug 25, 2021
BLS BASIC LIFE SUPPORT

How To Become a Philosophy Professor in 6 Steps (With Tips)

If you're curious about answering big life questions and discussing different viewpoints with others, becoming a philosophy professor may be a great career for you. Philosophy professors spend their time teaching, researching and completing administrative work at a college or university. Understanding the process to become this type of professor can help you determine if this is a career you are interested in pursuing. In this article, we discuss what a philosophy professor is, share some of their job duties and provide steps for how to become a philosophy professor.

A Ph. D., or Doctor of Philosophy, is a high-level degree earned after a period of three or more years of graduate-level study, culminating in the creation, submission, presentation and defense of a research dissertation

Bacterial Genetics

- an overview

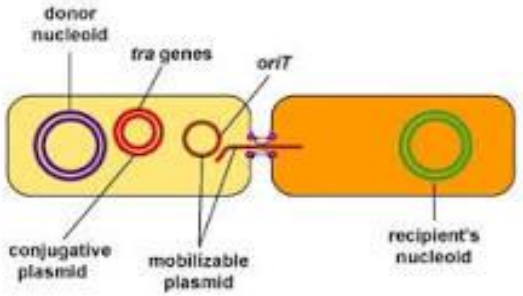
- | ScienceDirect Topics

Bacterial genetics

is the study of the mechanisms of heritable information in bacteria, their chromosomes, plasmids, transposons and phages.

Techniques that have enabled this discipline are culture in defined media, replica plating, mutagenesis, transformation, conjugation and transduction

Bacterial genetics is the study of how genetic information is transferred, either from a particular bacterium to its offspring or between interbreeding lines of bacteria, how genetic information is expressed, and how the genetic information (genotype) determines the physiology of the bacterium (phenotype).



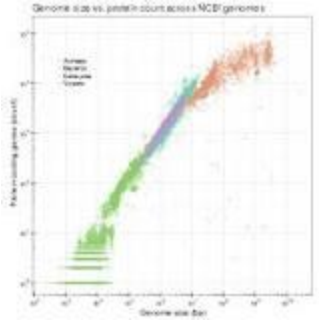
Search for: [What is bacterial genetics and its importance؟](#)

What are the ٣ types of bacterial gene transfer؟

There are three mechanisms of horizontal gene transfer in bacteria: transformation, transduction, and conjugation. Apr ٩, ٢٠٢٢

Who discovered bacterial genetics?

Transformation in bacteria was first observed in 1928 by Frederick Griffith and later (in 1944) examined at the molecular level by Oswald Avery and his colleagues who used the process to demonstrate that DNA was the genetic material of bacteria.



How many genes are in bacteria؟

Genome sequences show that parasitic bacteria have ٥٠٠–١٢٠٠ genes، Free-living bacteria have ١٥٠٠–٧٥٠٠ genes ، and archaea have ١٥٠٠–٢٧٠٠ genes.

الكائنات العتيقة أى بالغة من قديم الزمان الغابرينARCHAEA

What are the two types of bacteria?

Based on the structure of the cell wall, bacteria are classified into Gram-positive bacteria and Gram-negative bacteria.

What causes Genetic variation in bacteria? Prokaryotic cells have developed a number of methods for recombining their genetic material, which, in turn, contributes to their genetic diversity. The three most common ways that bacteria diversify their DNA are transformation, conjugation, and transduction.

How do bacteria gain genetic variation?

bacterial conjugation

increases genetic variation in prokaryotes by cells grabbing stray pieces of DNA from their environment or when cells directly exchange DNA (usually plasmids) with other cells; also known as genetic transfer.Feb 24, 2012

How are genes transferred between bacteria?

Ways for bacteria to share their genes:

Conjugation: Two bacteria can pair up and connect through structures in the cell membranes and then transfer DNA from one bacterial cell to another. Transduction: There are viruses called bacteriophages that can infect bacteria.

When DNA or genetic information is passed between bacteria?

Conjugation is the process by which one bacterium transfers genetic material to another through direct contact. During conjugation, one bacterium serves as the donor of the genetic material, and the other serves as the recipient. The donor bacterium carries a DNA sequence called the fertility factor, or F-factor.

بيقولواأن إقتران الأمشاج أو التزاوج أو تبادل التزواج أو تصريف الأفعال هو Conjugation

JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
28109091800516 - 2004578801966

What are the genetic elements of bacteria?

The genetic material of bacteria and plasmids is DNA. Bacterial viruses (bacteriophages or phages) have DNA or RNA as genetic material. The two essential functions of genetic material are replication and expression.

Who discovered bacterial genetics?

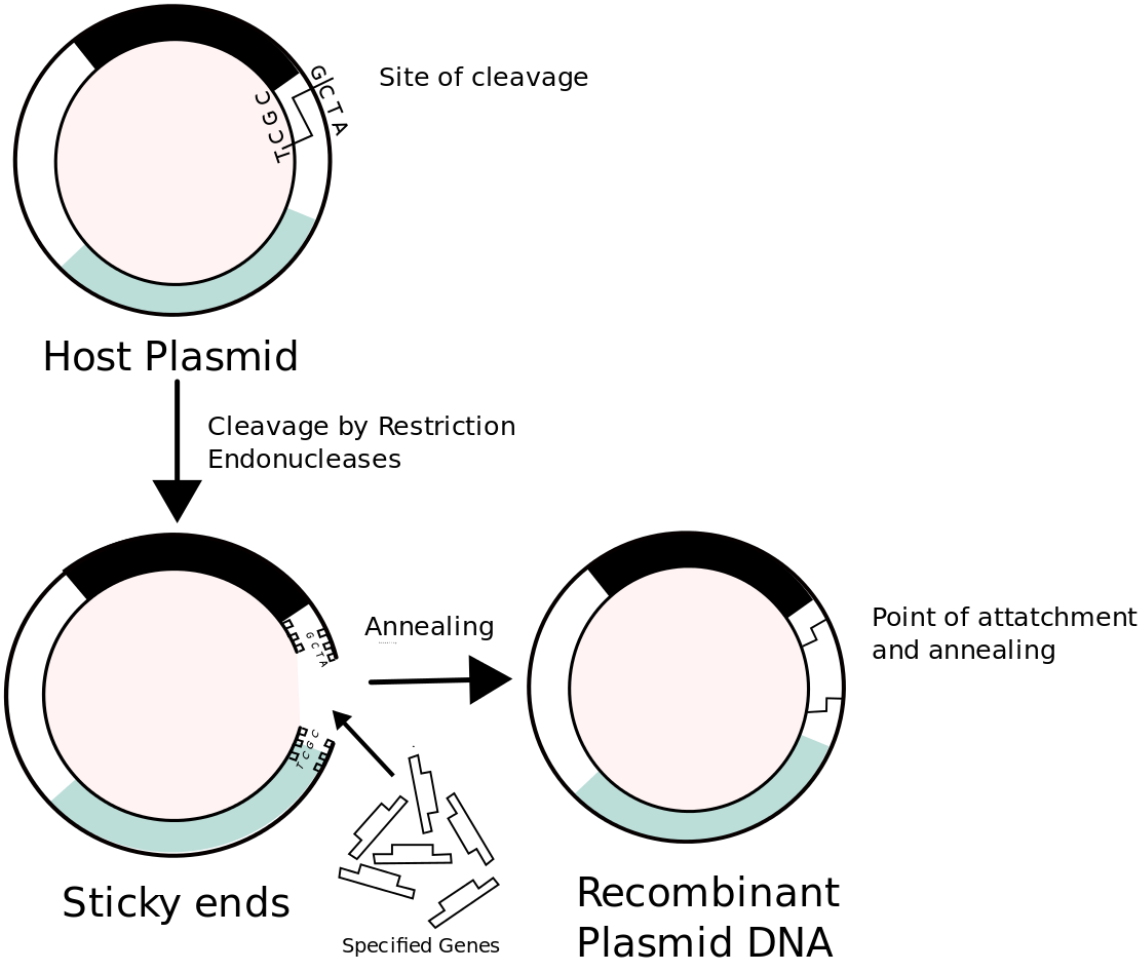
Transformation in bacteria was first observed in 1928 by Frederick Griffith and later (in 1944) examined at the molecular level by Oswald Avery and his colleagues who used the process to demonstrate that DNA was the genetic material of bacteria.

What is responsible for genetic variation in bacteria?

What is responsible for genetic variation in bacteria? - Mutations occur in the genome during binary fission. - Gene transfer from a different bacterial strain occurs. - Mutations produce new alleles, therefore new traits

What are the two forms that DNA takes in a bacterial cell?

bacterial plasmids and phages; with phages, the DNA fragments are inserted into phage DNA molecules. Each recombinant DNA molecule is put into a bacterial cell. حمض نووی معاد ترکیبه


G
C T A
T C G C
Site of cleavage
Host Plasmid
Cleavage by Restriction
Endonucleases
Annealing
Point of attatchment
and annealing
Sticky ends
Specified Genes
Recombinant
Plasmid DNA

How do bacteria take foreign DNA

Bacteria can take up foreign DNA in a process called transformation. Transformation is a key step in DNA cloning. It occurs after restriction digest and ligation and transfers newly made plasmids to bacteria. After transformation, bacteria are selected on antibiotic plates

Transformation تحويل وإنحراف وتعديل ونقل ومحول وتعميل وتصحيح وتطويروتقوية المناعة والمقاومة بأخذ أسباب الحياة للتأقلم مع الظروف البيئية الكونية .

DNA cloning من خلال دراساتى اللعب على عدد النيوكلوتيدات داخل الأحماض النووية الخلوية بقصها ولصقها من أجل المنفعة العامة وإن كان من يسوء إستخداماتها

DNA CLONING : FROM INTERNATIONAL INFORMATIONS COMMUNICATING WEBSITES NETWORKS معناها بالعربى

يعرف الاستنساخ التكاثري أو الجنسي بأنه إنتاج لكائن حي له نفس المادة الوارثية لكائن حي آخر المنسوخ منه.

Do bacteria have DNA or RNA?

The DNA of most bacteria is contained in a single circular molecule, called the bacterial chromosome. The chromosome, along with several proteins and RNA molecules, forms an irregularly shaped structure called the nucleoid. This sits in the cytoplasm of the bacterial cell.

How many chromosomes do bacteria have?

one chromosome

Most bacteria have a genome that consists of a single DNA molecule (i.e., one chromosome) that is several million base pairs in size and is "circular" (doesn't have ends like chromosomes of eukaryotic organisms)

What is bacterial DNA called?

A plasmid is a small circular DNA molecule found in bacteria and some other microscopic organisms. Plasmids are physically separate from chromosomal DNA and replicate independently.3 days ago

Where bacterial genes are located?

In addition to protein-structural genes (cistrons, which are coding genes), the bacterial chromosome contains genes for ribosomal and transfer ribonucleic acid (tRNA). Bacterial genes are often grouped intooperons or islands (e.g.,pathogenicity islands) that share function or coordinate their control.

CODING GENES

من خلال دراساتى الفعالةمنذ ١٩٨١

هى الجينات الحياتية الفعالةمنذ ١٩٨١

ترجمة و معنى genetic code في قاموس الكل عربي انجليزي

النص الأصلي	المعنى
كود وراثي [عامة]	genetic code
الشفرة الوراثية [عامة]	genetic code
الكود الجيني [عامة]	genetic code
شفرة جينية [عامة]	genetic code
شفرة جينية []	genetic code
الشفرة الوراثية []	genetic code
معجم الشفرات [كيمياء]	genetic code dictionary
الترميز المحافظ وراثيًا [عامة]	genetically conservative coding
مدونة قواعد السلوك الاختيارية بشأن إطلاق الكائنات الحية المحورة بيئياً [بيئة]	Voluntary Code of Conduct for the Release of Genetically

النص الأصلي	المعنى
الرَّاموزُ الجينِيّ [عامة]	genetic code
الكود الجيني [عامة]	genetic code
رامُوزُ الجينات [عامة]	genetic code
شفرة جينية [عامة]	genetic code
شفرة وراثية [عامة]	genetic code

What is bacterial genome called?

The nucleoid (meaning nucleus-like) is an irregularly-shaped region within the cell of a prokaryote that contains all or most of the genetic material, called as genophore.

Do bacteria have DNA?

In many bacteria the DNA is present as a single circular chromosome, although some bacteria may contain two chromosomes, and in some cases the DNA is linear rather than circular. A variable number of smaller, usually circular (though sometimes linear) DNA molecules, called plasmids, can carry auxiliary information.

bacteria - Genetic content | Britannica

https://www.britannica.com › science › Genetic-conten

Why does mutation occur in bacteria?

Before the bacterium can divide, it needs to make two identical copies of the DNA in its chromosome; one for each cell. Every time the bacterium goes through this process there is a chance (or risk, depending on the end result) that errors occur; so-called mutations ولكن من وجهة فكرى أن القادر على التحويلات الجينية كأساس للحياة لا يحدث به طفرات لأنها عيوب وراثية خلقية

Mutations and selection - ReAct - Action on Antibiotic Resistance

https://www.reactgroup.org

When DNA or genetic information is passed between bacteria?

Transduction

is the transfer of DNA from one bacterium to another by means of a bacteria-infecting virus called a bacteriophage.

Do all bacteria have plasmids?
Yes, Plasmids naturally exist in all bacterial cells. Plasmids are a small, circular, double-stranded DNA molecule, which is naturally found in all Bacterial cells. These plasmids are separated from chromosomal DNA and have the capability to replicate independently.

How many plasmids are in a bacterial cell?

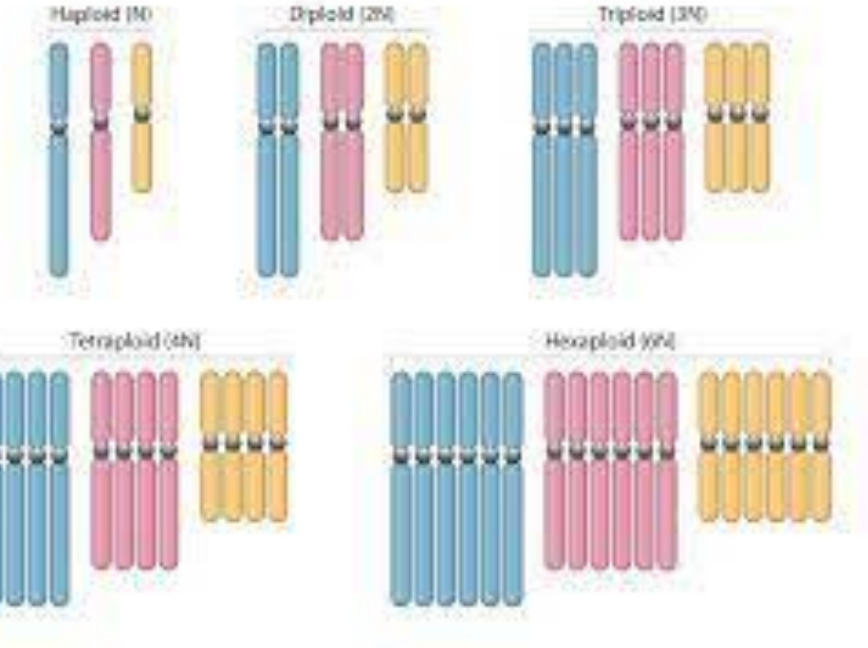
Most plasmids are circular, made of DNA, and much smaller than chromosomes. The copy number is the number of copies of the plasmid in each bacterial cell. For most plasmids, it is 1 or 2 copies per chromosome, but it may be as many as 50 or more for certain small plasmids such as the ColE plasmids.

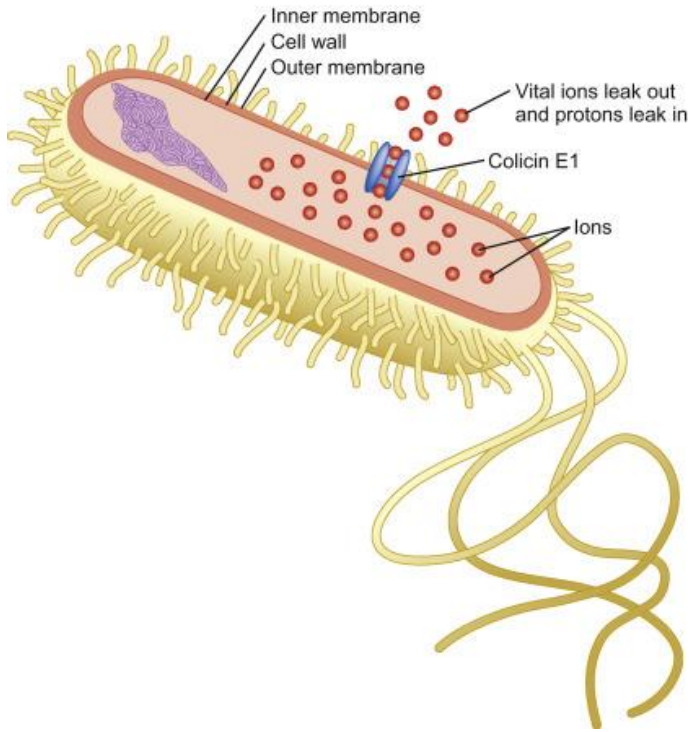
ColE1 is a plasmid found in bacteria. Its name derives from the fact that it carries a gene for colicin E1 (the cea gene). It also codes for immunity from this product with the imm gene. In addition, the plasmid has a series of mobility (mob) genes

Most Colicins Kill by One of Two Different Mechanisms

The Col plasmids allow the strains of *E. coli* that possess them to kill other related bacteria. There are two basic approaches to this. The first is to damage the victim's cell membrane. A gene on the ColE1 plasmid encodes the colicin E1 protein that inserts itself through the membrane of the target cell and creates a channel allowing vital cell contents, including essential ions, to leak out and protons to flood into the cell (Fig. 20.18).

The influx of protons collapses the proton motive force. The energy derived from the proton motive force drives the production of ATP and the uptake of many nutrients, without which the bacteria quickly die. A single molecule of colicin E1 that penetrates the membrane is enough to kill the target cell. Colicin I and colicin V operate by a similar mechanism.


Haploid (N)
Diploid (2N)
Triploid (3N)
Tetraploid (4N)
Hexaploid (6N)


Inner membrane
Cell wall
Outer membrane
Vital ions leak out
and protons leak in
Colicin E1
Ions

How are genes inserted into bacteria

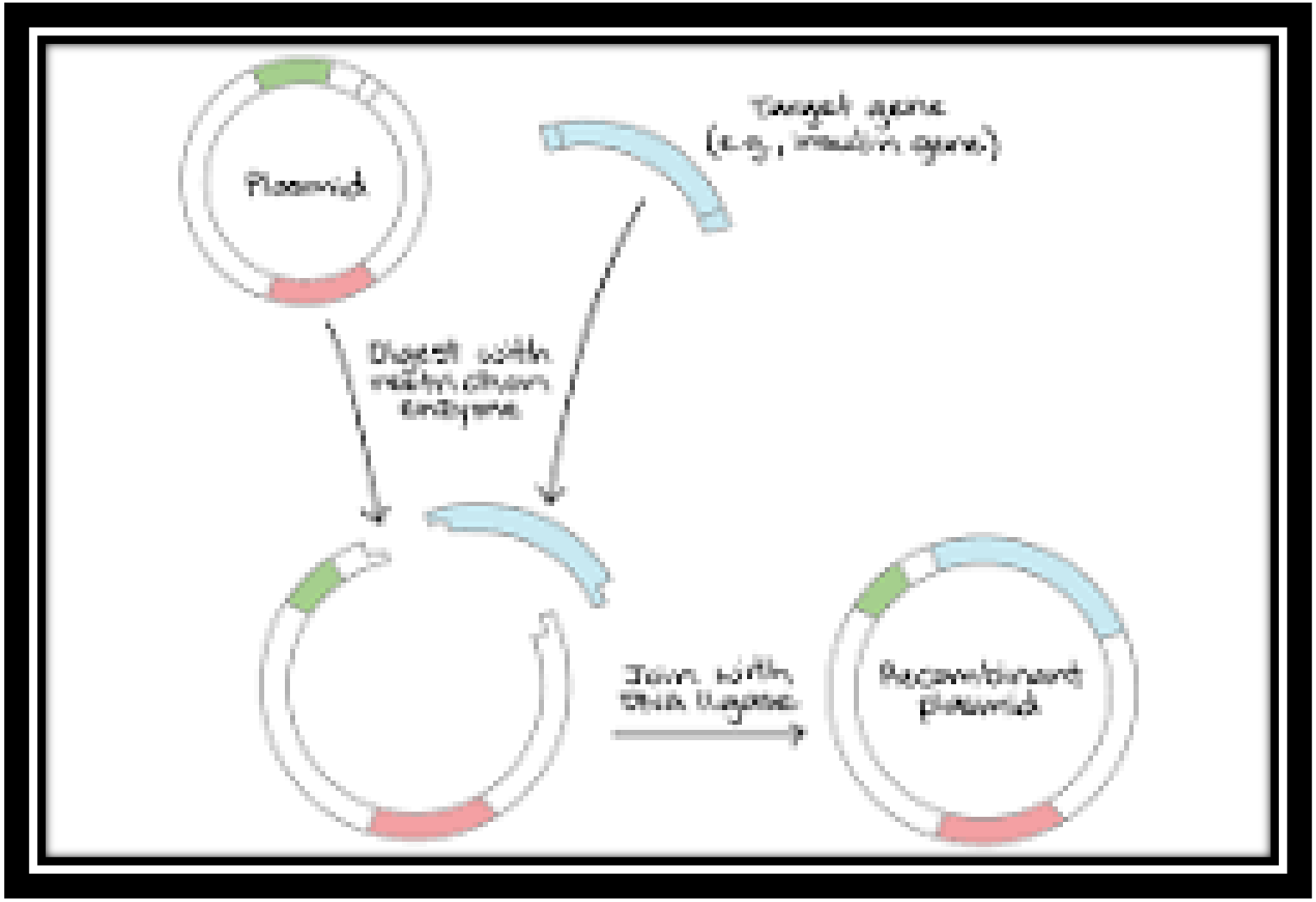
The basic steps are:

1.Cut open the plasmid and "paste" in the gene. This process relies on restriction enzymes (which cut DNA) and DNA ligase (which joins DNA.(

2.Insert the plasmid into bacteria... .

3.Grow up lots of plasmid-carrying bacteria and use them as "factories" to make the protein

Overview: DNA cloning (article) - Khan Academy


Plasmid
Target gene
(e.g., insulin gene)
Digest with
restriction
endonuclease
Join with
DNA ligase
Recombinant
plasmid

Is bacteria haploid or diploid?

haploid

Since bacteria are generally considered genetically haploid (see below), have a rapid generation time and can be easily grown to large population densities, traditional genetic analysis is that much more straightforward than for diploid eukaryotes Jan 1, 2001

Haploid Cells

أحادى الصيغة الصبغية-

Diploid Cells

ثنائى الصيغة الصبغية

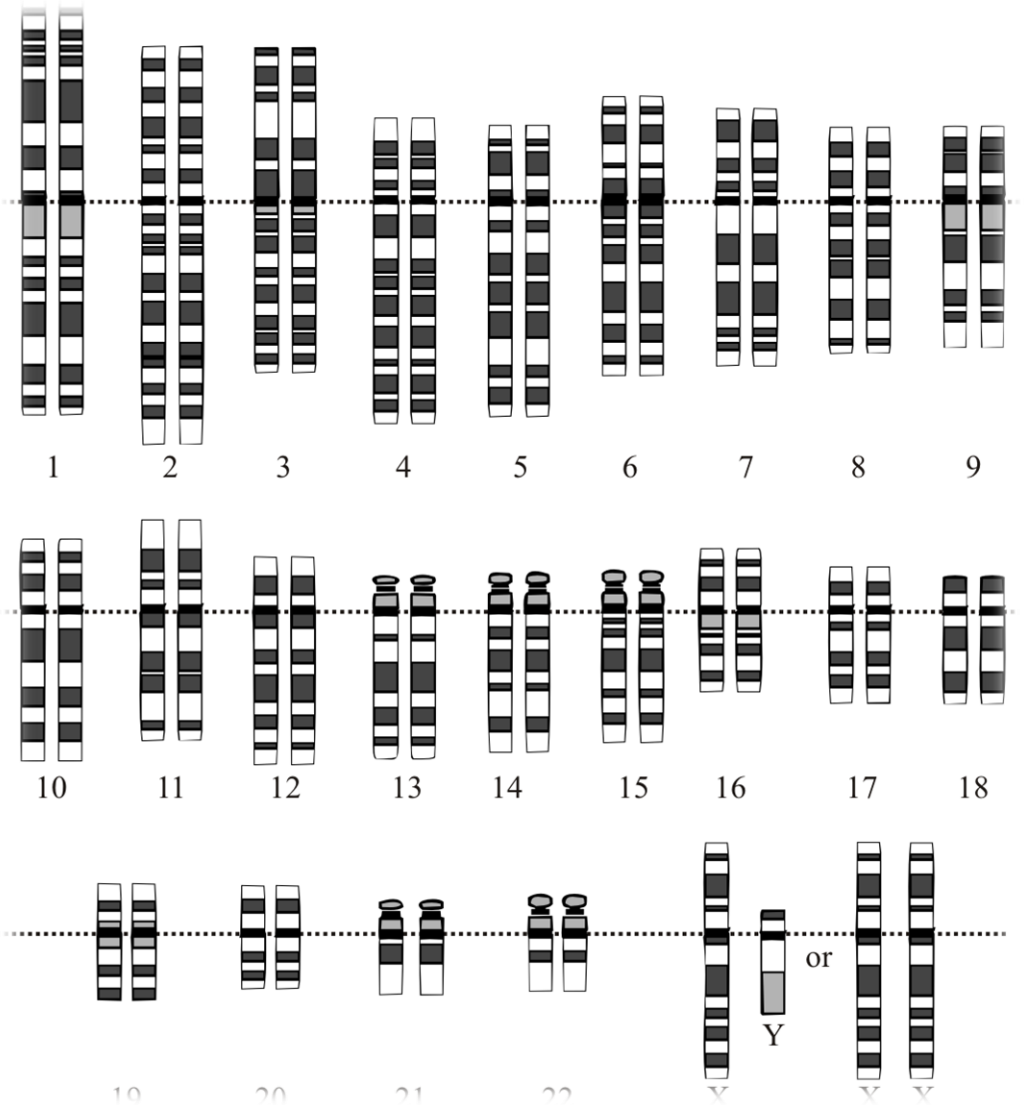
Triploid cells ثلاثى الصيغة الصبغية

Quadriloid cells رباعى الصيغة الصبغية,

Do bacteria have enzymes?
Bacteria are living cells which have the capabilities of consuming wastes of different types, reproducing, and actually producing enzymes. Better said, bacteria are the factories that produce enzymes.

Bacteria vs Enzymes vs Chemicals - UltraClear
https://ultraclear.com › bacteria

(monoploidوكذلك Haploidفي علم الأحياء، تطلق صفة **فرداني** (بالإنجليزية:
على الخلية التي تمتلك نصف العدد المعتاد للكروموسومات.[١][٢] توجد الكائنات
من حقيقيات النوى على شكل خلايا بصيغة صبغية هي الضعفانية، حيث تأتي مجموعة
من الكروموسومات من كل من الأبوين، لكن بعد عملية الانقسام الاختزالي سيصبح عدد
الكروموسومات في الأمشاج نصف العدد، وهي حالة الفرداني.
في الإنسان، العدد الضعفاني هو ٤٦ (٢×٢٣)، أما العدد الفرداني
في النطفة والبويضة فهو ٢٣.
بعض أنواع الحيوانات توجد على شكل فرداني، مثل غشائيات
الأجنحة (تضم النمل والنحل والزنبار). هذه الحالة الوراثية الخاصة تسمى فردانية
ضعفانية.
توجد بعض النباتات والحيوانات على شكل متعدد الصيغة الصبغية
حيث يوجد أكثر من زوجين من الكروموسومات، حيث (polyploidبالإنجليزية:
يوجد أحد أنواع القمح على شكل ٦ مجموعات من الكروموسومات.


1
2
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هذه الصُّورة هي عملٌ مِن إنتاج معاهد الصّحة الوطنية الأَمريكيَّة، وهي جزءٌ من وزارة الصّحة والخدمات البشريَّة في الولايات المُتحدة الأمريكيَّة. وبما أنَّها عملٌ من إنتاج الحكومة الاتحادية للولايات المُتحدة، فإنَّ هذه الصُّورة تقعُ في النِّطاق العامّ.

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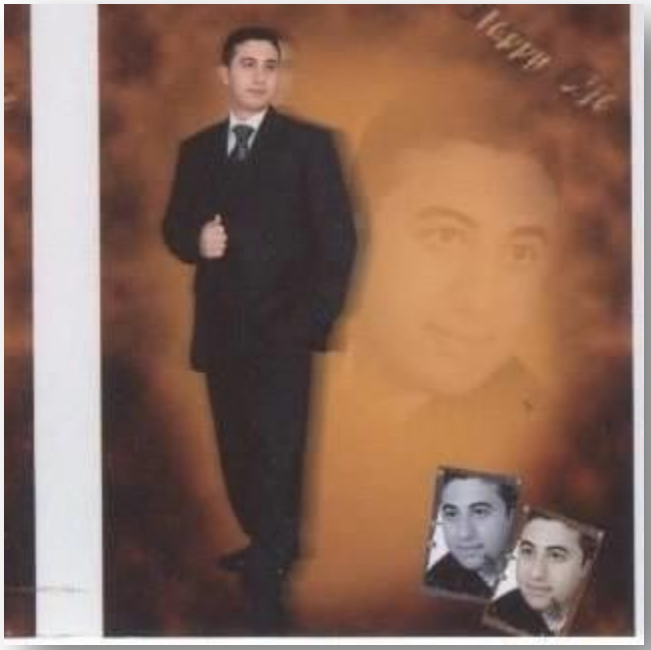
هذا الملفُّ مَلحُوظٌ بصفته غيرَ مُقيَّدٍ بحقوق التَّأليف والنشر، وهذا يشمل أيضاً الحقوق المُجاورة أَو ذات الصلة جميعُها.



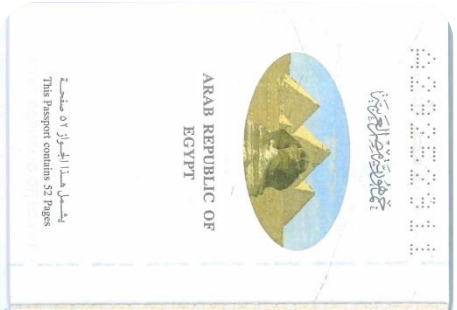
Bacteria

Bacteria, also called germs, are microscopic organisms not visible with the naked eye. Bacteria are everywhere, both inside and outside of your body.

Bacteria are small single-celled organisms. Bacteria are found almost everywhere on Earth and are vital to the planet's ecosystems. Some species can live under extreme conditions of temperature and pressure. The human body is full of bacteria, and in fact is estimated to contain more bacterial cells than human cells.

Bacteria are mostly free-living organisms often consisting of one biological cell. They constitute a large domain of prokaryotic microorganisms. Typically a few micrometres in length, bacteria were among the first life forms to appear on Earth, and are present in most of its habitats.

Most bacteria in the body are harmless, and some are even helpful. A relatively small number of species cause disease.


ARAB REPUBLIC OF EGYPT
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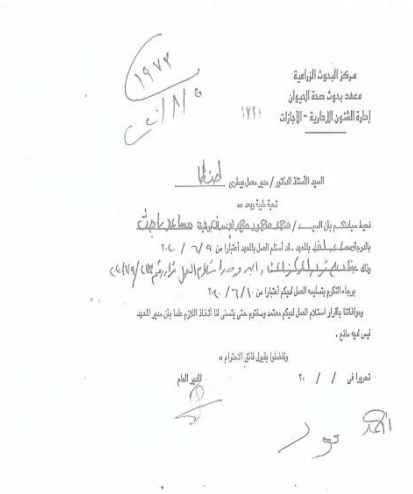
MOHAMMAD MAHMOUD MOHAMMAD YOUSSEF WAHIBA
09/09/1981 BENEIRA
EGYPTIAN M
10/11/2026
VETERINARIAN
P<EGYWAHIBA<<MOHAMMAD<MAHMOUD<MOHAMMAD<YOUSS
A292529113EGY8109091M2811109<<<<<<<<<<<<<<DO

جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتاريخ : ١٩٨١/٩/٩
الجنسية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٠٢١/٤/٢٧
تاريخ اعتماد مجلس الجامعة : ٢٠٢١/٥/١٩
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بقرار رقم ١٦٩ بتاريخ ٢٠٢٠/١٠/٥
المختص رئيس القسم وكيل الكلية للدراسات العليا عميد الكلية
مستخرج من بيانات مركز الشهادات الموحدة بتاريخ ٢٠٢١/٦/٢٠

جامعة
كفر الشيخ
شهادة تخرج
063342
تشهد كلية الطب البيطري
أن / محمد محمود محمد يوسف
المولود فى البحيرة بتاريخ ١٩٨١م
قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية
فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً
مجموع تراكمى ٢٤٧٥ / ٣٣٠٠ النسبة المئوية ٧٥,٠٠%
وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م
ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا
الموظف المسئول المراجع مدير شئون الطلاب عميد الكلية
تحريراً فى ٢٠١٢/٥/٢٢م


مركز البحوث الزراعية
معمل بحوث صحة الحيوان
إجازة الشئون الإدارية - الإجازات
السيد الأستاذ الدكتور / مدير معمل بيطرى
نحية طيبة وبعد ،،
يرجاء التكرم بتشابه العمل لديكم اختيارا من ٢٠ / ٦ / ٢٠
وبرفائنا بالقرار استلام العمل لديكم منتدبا وبستحوم حتى يتسنى لنا أخذذ القرار فضا بان مدير العديد
ليس لديه مانع .
وتفضلوا بقبول فائق الاحترام ،،
تحريراً فى / / ٢٠
المدير العام

جمهورية مصر العربية
بطاقة تحقيق الشخصية
محمد
محمود محمد يوسف وهيبه
ش العوض -كفر الزيات
مركز كفر الزيات - الغربية
٢٨١٠٩٠٩١٨٠٠٥١٦
HQ2270868

٢٨١٠٩٠٩١٨٠٠٥١٦ ٢٠١٨/٠٧
طبيب بيطرى
معهد بحوث صحة الحيوان بطنطا
ذكر مسلم أعزب
البطاقة سارية حتى ٢٠٢٥/٠٧/٢٣
٣

وحده مرور دمنهور
رخصه قياده خاصه
١١٣٦٢٢
جمهورية مصر العربية
وزارة الداخلية
مرور البحيره
محمد محمود
محمد يوسف
مصرى
بكالوريوس طب بيطرى
ش طور سيناء ش الشيخة
دمنهور.

Morphology

Bacteria are complex and highly variable microbes. They come in four basic shapes: spherical (cocci), rod-shaped (bacilli), arc-shaped (vibrio), and spiral (spirochete)

Cell wall

Eubacteria are enclosed by a cell wall. The wall is made of cross-linked chains of peptidoglycan, a polymer that combines both amino acid and sugar chains. ... Some types of bacteria have an additional layer outside the cell wall. This layer is made from lipopolysaccharide (LPS), a combination of lipids and sugars.

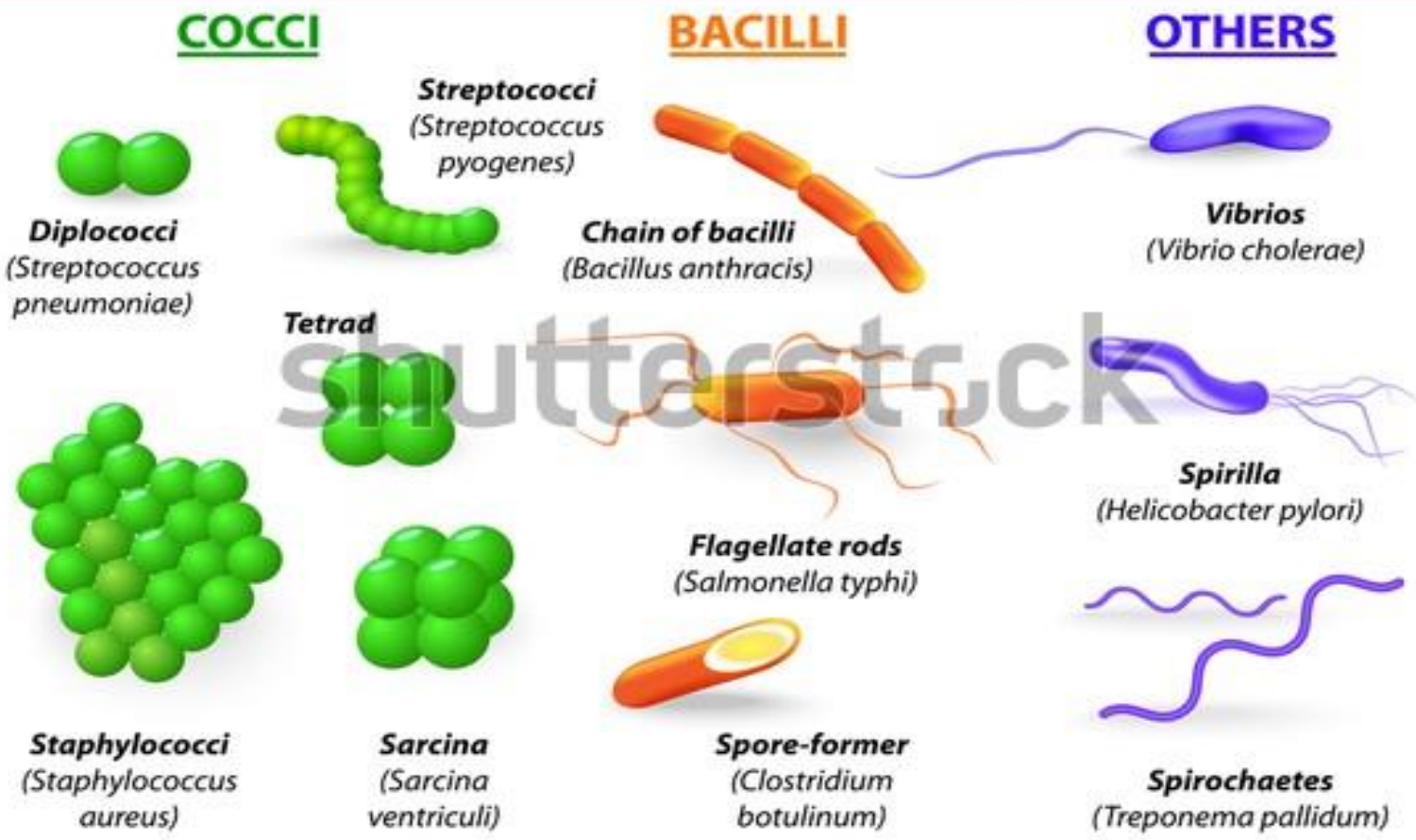
Nutrition

Nutrition. A great many of the most familiar eubacteria are heterotrophs, meaning they must take food in from outside sources. Of the heterotrophs, the majority are saprophytes, which consume dead material, or parasites, which live on or within another organism at the host's expense.

Treatment

Antibiotics are the usual treatment. When you take antibiotics, follow the directions carefully. Each time you take antibiotics, you increase the chances that bacteria in your body will learn to resist them causing antibiotic resistance. Later, you could get or spread an infection that those antibiotics cannot cure.

SHAPES OF BACTERIA



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SHAPES OF BACTERIA

SPHERICAL (COCCI)

Streptococcus pneumoniae

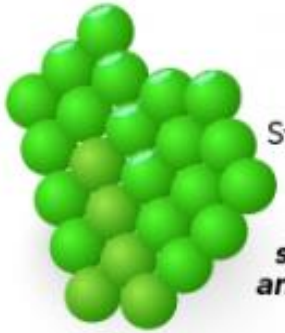
causes Pneumonia

Streptococcus pyogenes

causes Strep throat

Micrococcus luteus

causes armpits to stink



Staphylococcus aureus

can cause sinus infections and food poisoing

ROD-SHAPED (BACILLI)

(Bacillus anthracis)

causes Anthrax

(Salmonella enterica)

causes Typhoid

(Clostridium botulinum)

causes Botulism

SPIRAL-SHAPED (AND OTHERS)

(Vibrio cholerae)

causes Cholera

(Helicobacter pylori)

can cause stomach ulcers

(Treponema pallidum)

causes Syphilis

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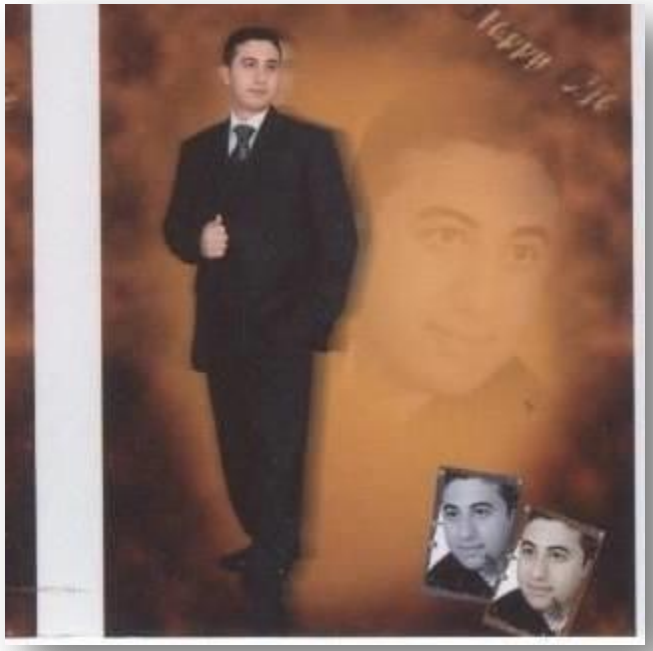


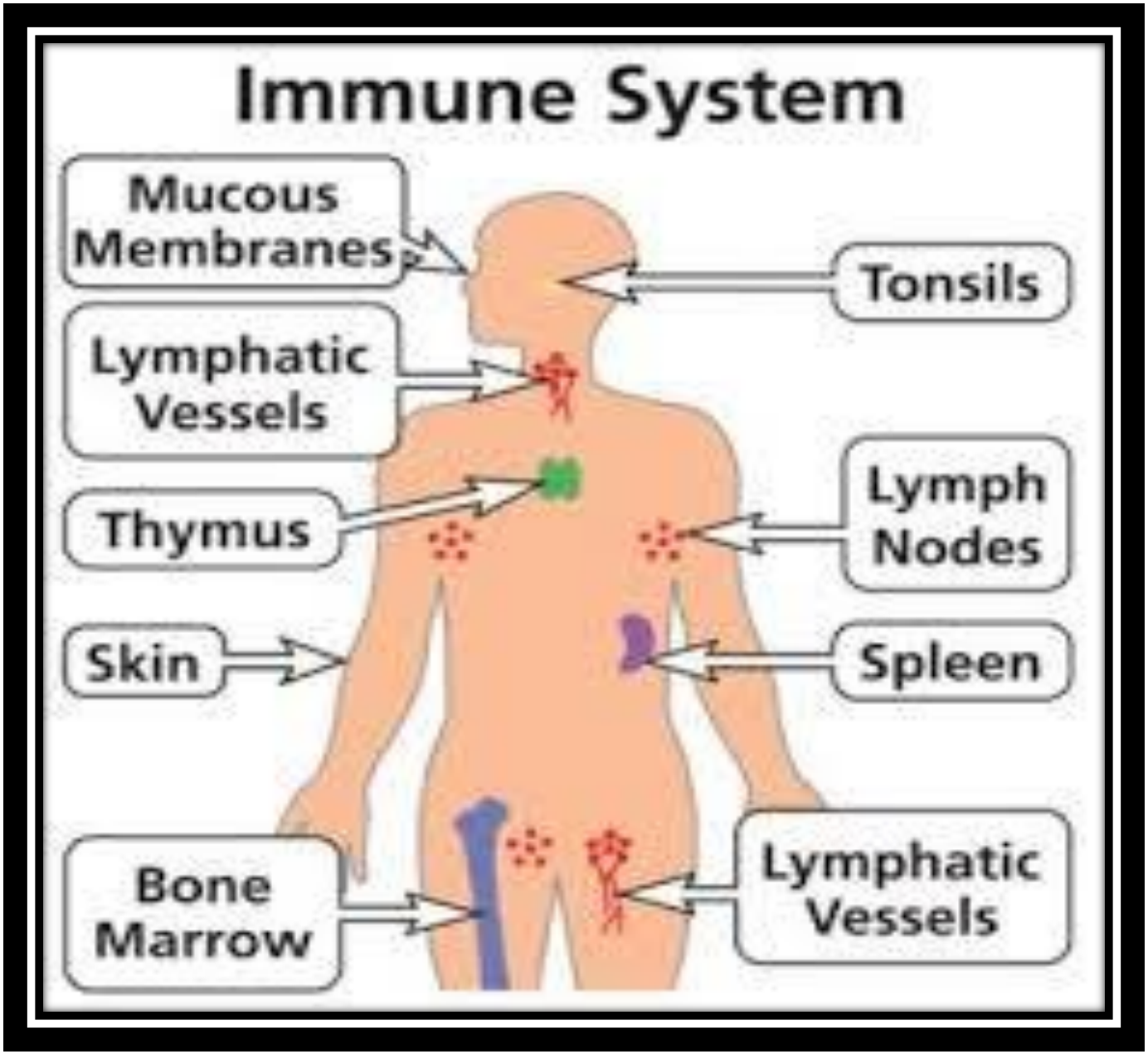
immune system

What is immune system and its function

Your immune system is a large network of organs, white blood cells, proteins (antibodies) and chemicals. This system works together to protect you from foreign invaders (bacteria, viruses, parasites, and fungi) that cause infection, illness and disease.

the organs and processes of the body that provide resistance to infection and toxins. Organs include the thymus, bone marrow, and lymph nodes.

What is the immune system? The immune system protects your child's body from outside invaders. These include germs such as bacteria, viruses, and fungi, and toxins (chemicals made by microbes). The immune system is made up of different organs, cells, and proteins that work together.


Immune System
Mucous Membranes
Tonsils
Lymphatic Vessels
Thymus
Lymph Nodes
Skin
Spleen
Bone Marrow
Lymphatic Vessels

What are the 4 immune systems?

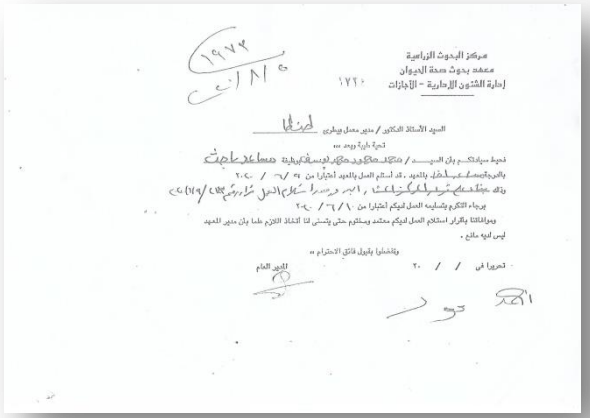
What are the 4 immune systems?
The main parts of the immune system are: white blood cells, antibodies, the complement system, the lymphatic system, the spleen, the thymus, and the bone marrow. These are the parts of your immune system that actively fight infection.

There are 2 main parts of the immune system:

1-The innate immune system. You are born with this.

2-The adaptive immune system. You develop this when your body is exposed to microbes or chemicals released by microbes.

These 2 immune systems work together.



The innate immune system

This is your child's rapid response system. It is the first to respond when it finds an invader. It is made up of the skin, the eye's cornea, and the mucous membrane that lines the respiratory, gastrointestinal, and genitourinary tracts. These all create physical barriers to help protect your child's body. They protect against harmful germs, parasites (such as worms), or cells (such as cancer). The innate immune system is inherited. It is active from the moment your child is born. When this system recognizes an invader, it goes into action right away. The cells of this immune system surround and cover the invader. The invader is killed inside the immune system cells (called phagocytes).

The acquired immune system

The acquired immune system, with help from the innate system, makes special proteins (called antibodies) to protect your body from a specific invader. These antibodies are developed by cells called B lymphocytes after the body has been exposed to the invader. The antibodies stay in your child's body. It can take several days for antibodies to form. But after the first exposure, the immune system will recognize the invader and defend against it. The acquired immune system changes during your child's life. Immunizations train your child's immune system to make antibodies to protect them from harmful diseases.

The cells of both parts of the immune system are made in different organs of the body, including:

Adenoids. Two glands located at the back of the nasal passage.
Bone marrow. The soft, spongy tissue found in bone cavities.
Lymph nodes. Small organs shaped like beans, which are located all over the body and connect via the lymphatic vessels.
Lymphatic vessels. A network of channels all over the body that carries lymphocytes to the lymphoid organs and bloodstream.
Peyer patches. Lymphoid tissue in the small intestine.
Spleen. A fist-sized organ located in the belly (abdominal) cavity.
Thymus. Two lobes that join in front of the windpipe (trachea) behind the breastbone.
Tonsils. Two oval masses in the back of the throat.

THE TOPIC TODAY

How do vaccines work?

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

Germs are all around us, both in our environment and in our bodies. When a person is susceptible and they encounter a harmful organism, it can lead to disease and death.
The body has many ways of defending itself against pathogens (disease-causing organisms). Skin, mucus, and cilia (microscopic hairs that move debris away from the lungs) all work as physical barriers to prevent pathogens from entering the body in the first place.
When a pathogen does infect the body, our body's defences, called the immune system, are triggered and the pathogen is attacked and destroyed or overcome.

The body's natural response

A pathogen is a bacterium, virus, parasite or fungus that can cause disease within the body. Each pathogen is made up of several subparts, usually unique to that specific pathogen and the disease it causes.

The subpart of a pathogen that causes the formation of antibodies is called an antigen.

The antibodies produced in response to the pathogen’s antigen are an important part of the immune system.

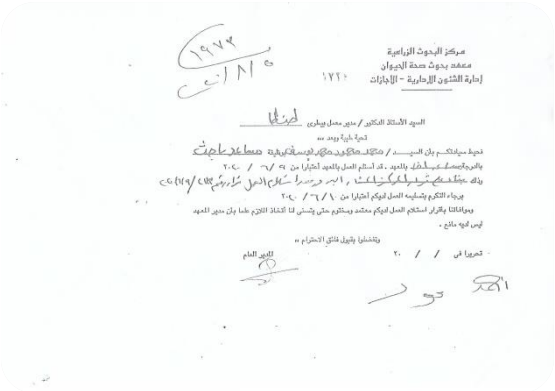
You can consider antibodies as the soldiers in your body's defense system.

Each antibody, or soldier, in our system is trained to recognize one specific antigen.

We have thousands of different antibodies in our bodies.

When the human body is exposed to an antigen for the first time, it takes time for the immune system to respond and produce antibodies specific to that antigen.

In the meantime, the person is susceptible to becoming ill.



مركز البحوث الزراعية
معهد بحوث صحة الحيوان
إدارة الشئون الإدارية - الإجازات

السيد الأستاذ الدكتور / مدير معمل بيطري

تحية طيبة وبعد ،،

تحيط سيادتكم بأن السيد / [illegible] بوسطة بقرية [illegible] مطاعجة
والمرفق بصحبته عبارة عن [illegible] بالمعهد ، قد استلم العمل بالمعهد اعتبارا من ٢٠٢٠/٦/٩
بناء عليه نرجو [illegible] ، [illegible] ١٩٧٢/٥٢ بتاريخ ٢٠٢٠/٦/٩

يرجاء التكرم بتأشيره العمل ليكم اعتبارا من ٢٠٢٠/٦/١٠
وهذا بالإقرار استلم العمل ليكم معتمد وبختوم حتى يتسنى لنا اتخاذ اللازم علما وان مدير المعهد
ليس فيه مانع .

وتفضلوا بقبول فائق الاحترام ،،

تحريرا في / / ٢٠

المدير العام

جامعة بنها

شهادة مؤقتة

تشهد كلية الطب البيطري

منح السيدة : محمد محمود محمد يوسف

المولودة في : البحيرة بتاريخ : ٢٩/٩/١٩٩٢

الجنسية : مصرى الرقم القومي : ٢٩٢٠٩٠٩١٨٠٠٢٦

درجة الماجستير في العلوم الطبية البيطرية تخصص:

"البكتريولوجيا والمناعة والفطريات"

وموضوع الرسالة: تواجد ميكروبات التسمم الغذائي في بعض أنواع اللحوم الطازجة في السوق المحلي.

تاريخ اعتماد مجلس الكلية : ٢٠٢١/٤/٢٧

تاريخ اعتماد مجلس الجامعة : ٢٠٢١/٥/١٩

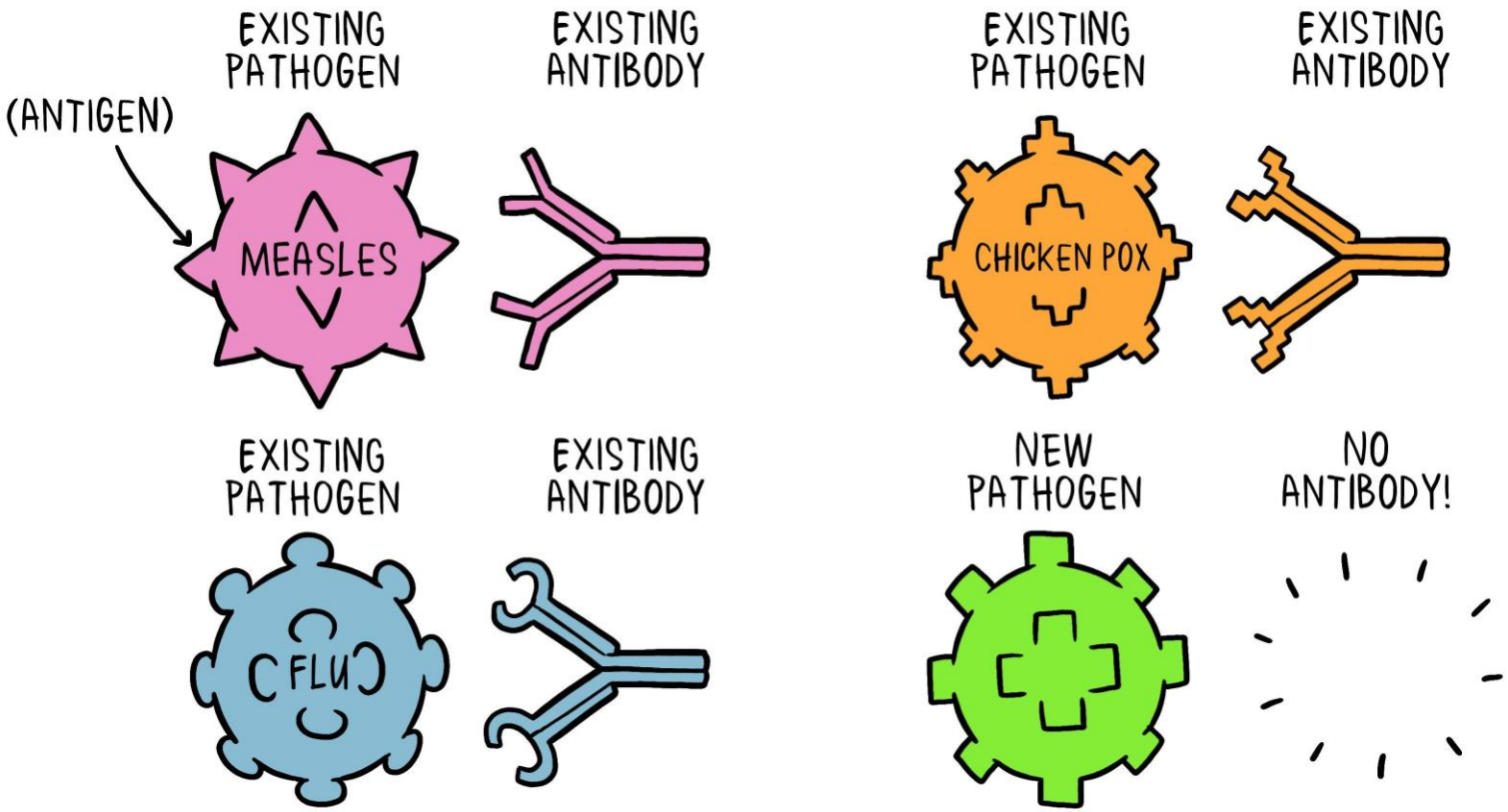
Once the antigen-specific antibodies are produced, they work with the rest of the immune system to destroy the pathogen and stop the disease.

Antibodies to one pathogen generally don't protect against another pathogen except when two pathogens are very similar to each other, like cousins.

Once the body produces antibodies in its primary response to an antigen, it also creates antibody-producing memory cells, which remain alive even after the pathogen is defeated by the antibodies.

If the body is exposed to the same pathogen more than once, the antibody response is much faster and more effective than the first time around because the memory cells are at the ready to pump out antibodies against that antigen.

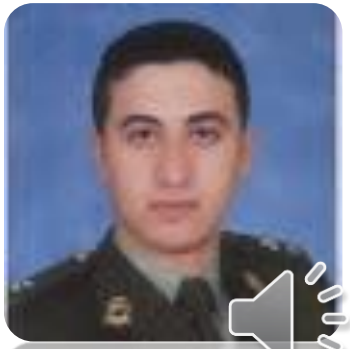
This means that if the person is exposed to the dangerous pathogen in the future, their immune system will be able to respond immediately, protecting against disease.



When a new pathogen or disease enters our body, it introduces a new antigen. For every new antigen, our body needs to build a specific antibody that can grab onto the antigen and defeat the pathogen.

How vaccines help

Vaccines contain weakened or inactive parts of a particular organism (antigen) that triggers an immune response within the body.



Newer vaccines contain the blueprint for producing antigens rather than the antigen itself.

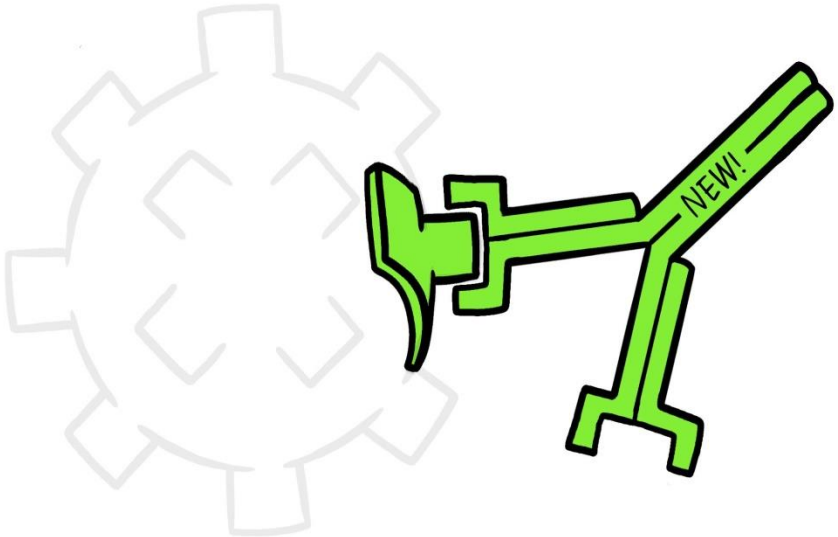
Regardless of whether the vaccine is made up of the antigen itself or the blueprint so that the body will produce the antigen, this weakened version will not cause the disease in the person receiving the vaccine,

but it will prompt their immune system to respond much as it would have on its first reaction to the actual pathogen.

.MOHAMMED

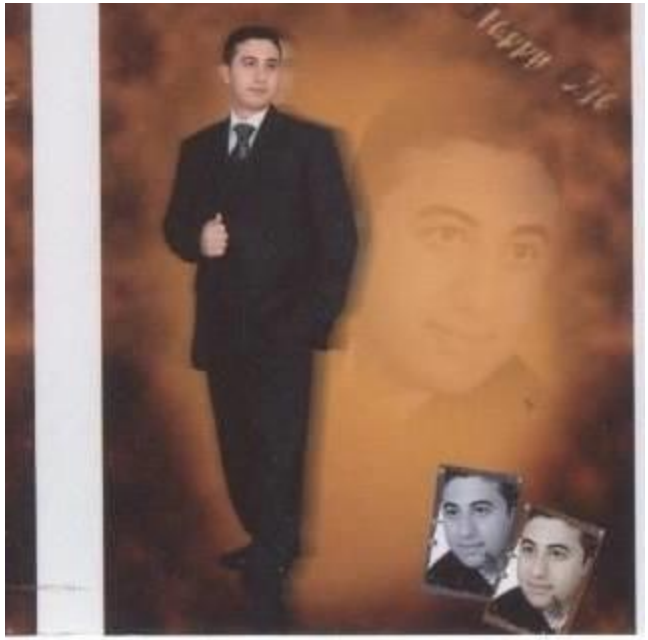
VACCINE

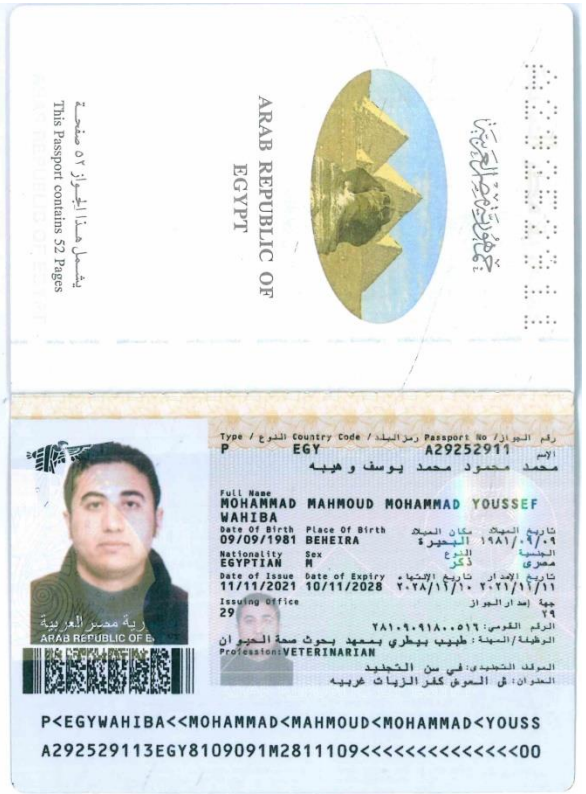
NEW ANTIBODY



A VACCINE is a tiny weakened non-dangerous fragment of the organism and includes parts of the antigen. It's enough that our body can learn to build the specific antibody. Then if the body encounters the real antigen later, as part of the real organism, it already knows how to defeat it.

Some vaccines require multiple doses, given weeks or months apart. This is sometimes needed to allow for the production of long-lived antibodies and development of memory cells. In this way, the body is trained to fight the specific disease-causing organism, building up memory of the pathogen so as to rapidly fight it if and when exposed in the future.





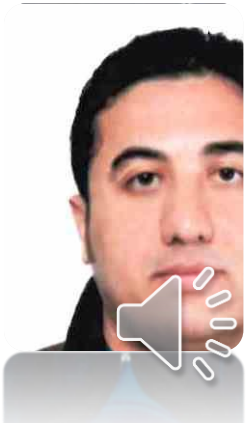
Herd immunity

When someone is vaccinated, they are very likely to be protected against the targeted disease.

But not everyone can be vaccinated. People with underlying health conditions that weaken their immune systems (such as cancer or HIV) or who have severe allergies to some vaccine components may not be able to get vaccinated with certain vaccines.

These people can still be protected if they live in and amongst others who are vaccinated.

When a lot of people in a community are vaccinated the pathogen has a hard time circulating because most of the people it encounters are immune.

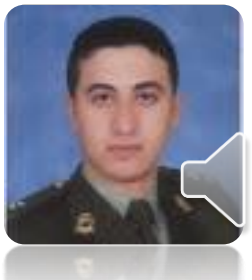


COMMUNITY VACCINATION PROTECTS THE WHOLE COMMUNITY EVEN THOSE WHO CAN NOT VACCINATED

This is called herd immunity.

Throughout history, humans have successfully developed vaccines for a number of life-threatening diseases, including meningitis, tetanus, measles and wild poliovirus.

In the early 1900s, polio was a worldwide disease, paralysing hundreds of thousands of people every year. By 1950, two effective vaccines against the disease had been developed. But vaccination in some parts of the world was still not common enough to stop the spread of polio, particularly in Africa. In the 1980s, a united worldwide effort to eradicate polio from the planet began.



Over many years and several decades, polio vaccination, using routine immunization visits and mass vaccination campaigns, has taken place in all continents. Millions of people, mostly children, have been vaccinated and in August 2020, the African continent was certified wild poliovirus free, joining all other parts of the world except Pakistan and Afghanistan, where polio has not yet been eradicated.

(SPONDYLOSIS)

(داء الفقار) (خشونة الفقرات)

مساعدباحث /محمدمحمودمحمديوسف وهيبه

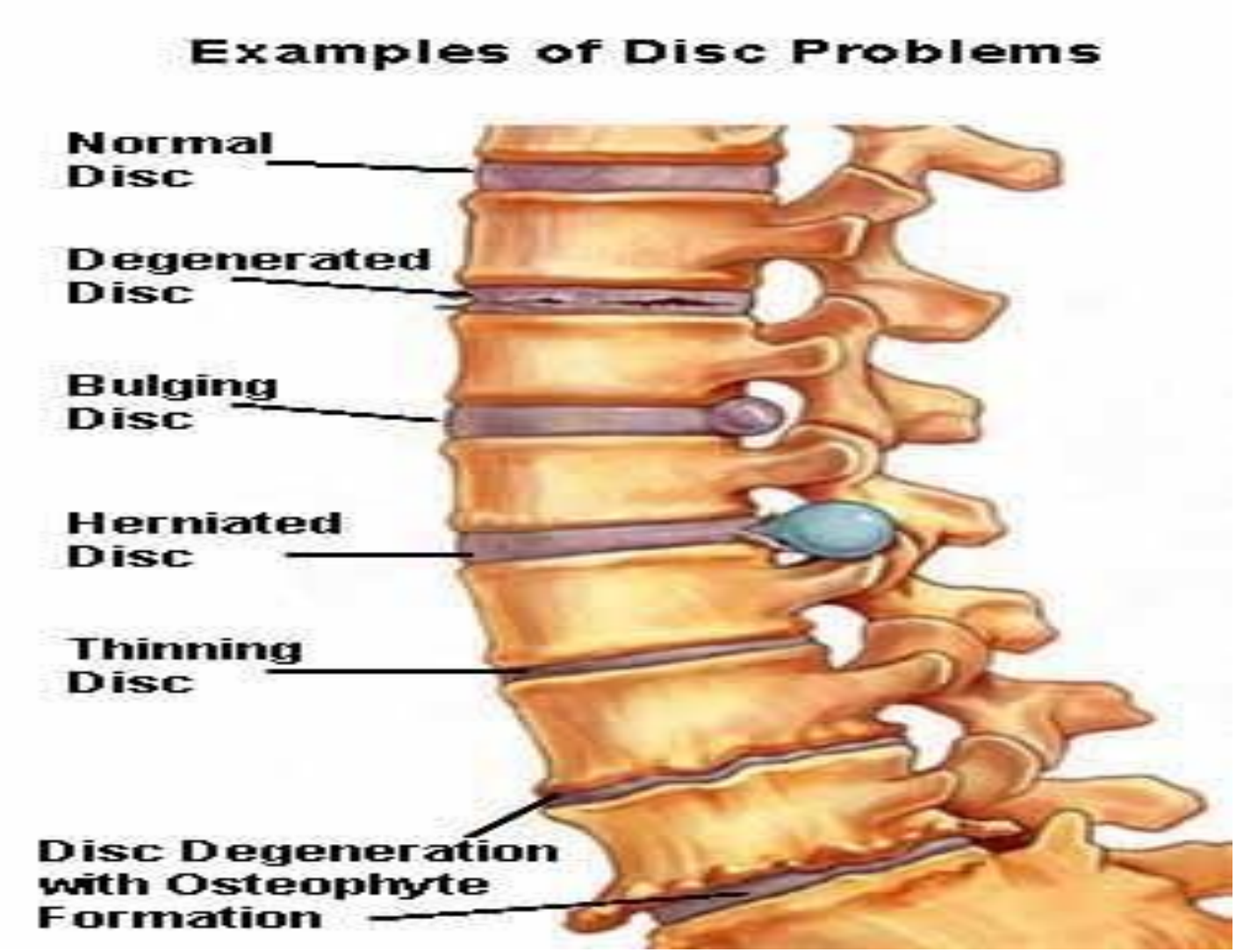
معهدبحوث الصحة الحيوانية

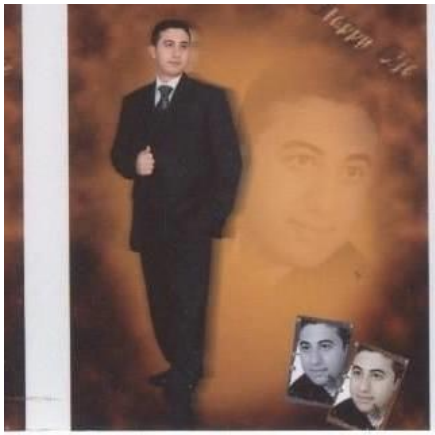
مركزالبحوث الزراعية

وزارة الزراعة

جمهورية مصرالعربية

يُعد داء الفقار الرقبية (خشونة الفقرات) مصطلحًا عامًا يصف التلف والتمزق المرتبط بالمرحلة العمرية والذي يصيب أقراص العمود الفقري في الرقبة.
وتظهر علامات الفُصال العظمي كلما أصاب الجفاف والانكماش الأقراص، بما يتضمن النتوءات العظمية على طول أطراف العظام. ويُعد داء الفقار الرقبية منتشرًا للغاية ويتفاقم مع التقدم في العمر.

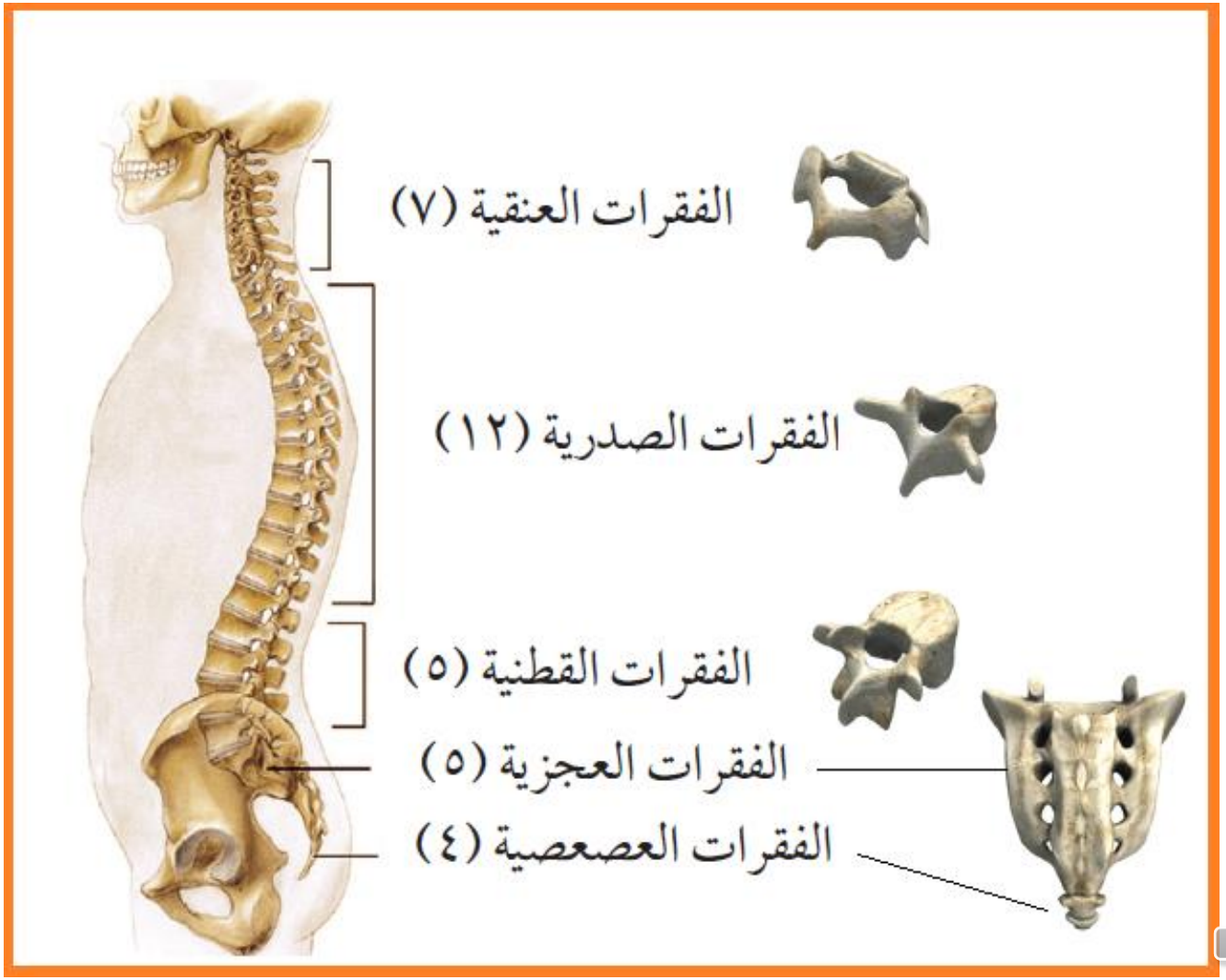

Examples of Disc Problems
Normal Disc
Degenerated Disc
Bulging Disc
Herniated Disc
Thinning Disc
Disc Degeneration with Osteophyte Formation



الفصال العظمى

ويُعرَف الالتهاب المفصلي العظمي في أغلب الحالات بالتآكل والاهتراء الناجمين عن استخدام المفصل بمرور الزمن. ولا يستهدف الالتهاب المفصلي العظمي النسيج الغضروفي وحسب، بل يؤثر كذلك على المفصل بالكامل. ويسبب تغيرات في العظام وتآكل الأنسجة الضامة التي تربط المفصل معًا وتربط العضلات بالعظام.

العمود الفِقْرِي أو العمود الفَقْرِي أو الفَقَار للكائنات الحية الفقارية مكون من سلسلة فِقْرَات مفرغة لحفظ النخاع الشوكي الذي يمتد فيها ويبلغ عدد الفِقْرَات عند الإنسان ٣٣ فقرة. يدعى الفراغ الذي يسكنه النخاع الشوكي بالنفق الفقري.


الفقرات العنقية (٧)
الفقرات الصدرية (١٢)
الفقرات القطنية (٥)
الفقرات العجزية (٥)
الفقرات العصعصية (٤)

لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة. اليكم لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة تشكل القسم السفلي من العمود الفقري (العجز والعصعص).

يقسم العمود الفقري إلى المناطق التالية:

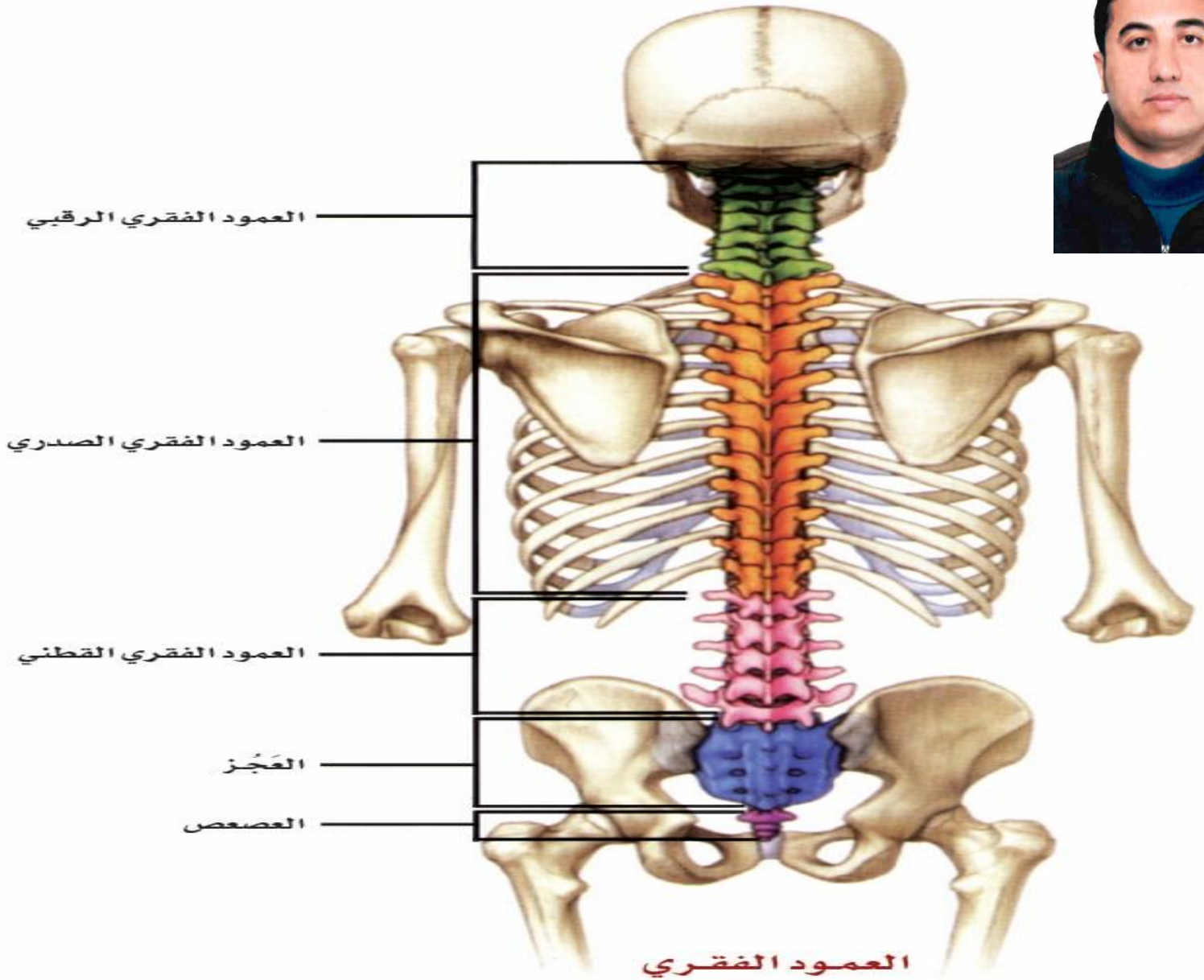
١. الفقرات الرقبية وعددها ٧ فقرات تشكل الانحناء الرقبي للعمود الفقري حيث تنحني هذه الفقرات بشكل طبيعي للأمام.

٢. الفقرات الظهرية أو العمود الفقري الصدري وعددها ١٢ فقرة ويكون انحناء العمود الفقري في هذه المنطقة نحو الخلف، وتتصل الفقرات الظهرية مع الأضلاع التي تكوّن القفص الصدري.

٣. الفقرات القطنية وعددها ٥ فقرات وهي تشكل منطقة أسفل الظهر ويكون انحناء العمود الفقري في هذه المنطقة للأمام بنفس جهة انحناء العمود الرقبي.

٤. العجز المكون من ٥ فقرات ملتحمة مع بعضها مشكلة قطعة عظمية واحدة مثلثية الشكل تقع تحت العمود الفقري القطني مباشرة.

٥. العصعص ويتكون من ٤ أو ٥ فقرات ملتحمة وهو القسم الانتهائي من العمود الفقري.



العمود الفقري

بيانات هامة للإتصال بصاحب

الموبايل نوكيا 130 –

دكتورمحمدمحمودوهيبه وعائلته :

دكتورمساعدباحث

/محمدمحمودمحمديوسف وهيبه

01210973074

أبى 01221868424

أمى 01206372319

أختى 01220781971

أخى 01288849405

دكتور عادل عبدالعزيز مدير العمل

01006646670

جامعة سوهاج
كلية الطب البيطري
لضمان جودة التعليم والاعتماد
لتدعيم الطلاب

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١ م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس في العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جداً) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

بيان التقديرات الحامل عليها الثناء، مراعاته بالكلية :-

الفرقة الأولى: دور: مايو عام: ١٩٩٨/١٩٩٩م

المادة	الفيزياء: العيوبة	الميكانيكا: ميكانيكا وصوتيات	الكيمياء: العامة	التشريح والأنسجة	المستويات	الوراثة والخدمة	أحياء: عيوب	لغة إنجليزية	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	ممتاز	جيد//	ممتاز	جيد جداً
الدرجة	٣٥	٧٦	٨٢	٧٧	٧٤	٩٤	٢٣	٤٥	٥١٦

الفرقة الثانية: دور: مايو عام ١٩٩٩/٢٠٠٠م

المادة	تشريح الطيور ومكارن	الفسيولوجيا	طفيليات: عمومية	تغذية الحيوان والدواجن وأمراض سوء التغذية	سلوكيات الحيوان والدواجن وبر دواجنها	خاص الفم	التقدير العام
التقدير	جيد جداً	جيد جداً	ممتاز	جيد//	مقبول	مقبول	جيد جداً
الدرجة	٨١	٨٢	٩١	٧٢	٦٤	٣٠	٣٨٩

الفرقة الثالثة: دور: مايو عام: ٢٠٠٠/٢٠٠١م

المادة	باثولوجيا عام	مختبرباثولوجيا وطفيليات البعامة	التطفيليات	فارماكولوجيا	إنعاش حيواني	القضاء وإدارة برام م	فيروليوجيا	التقدير العام
التقدير	مقبول	جيد جداً	جيد جداً	مقبول	جيد//	جيد//	مقبول	جيد//
الدرجة	٥٠	٨١	٧٨	٧١	٦٩	٧٤	٣٢	٤٥٥

الفرقة الرابعة: دور: مايو عام: ٢٠٠١/٢٠٠٢م

المادة	باثولوجيا تشريحيه مرضي	أمراض باطنة	جراحة عامة (تناسلية)	جراحة عامة وتخدير	باثولوجيا وطب شرعي	الرقابة الصحية على الألبان	باثولوجيا إكلينيكية	أمراض التناسلية	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	جيد//	جيد//	جيد جداً	جيد//
الدرجة	٧٤	٦٩	٧٧	٧٦	٧٠	٧٣	٣٥	٤٠	٥١٤

الفرقة الخامسة: دور: مايو عام ٢٠٠٢/٢٠٠٣م

المادة	أمراض باطنه	جراحة عامة والتخدير	التوليد والتلقيح الصناعي	أمراض تناسلية	أمراض الطيور والأرانب	الرقابة الصحية على اللحوم ومنتجاتها	صحة الحيوان والدواجن والبيئة	الأمراض المشتركة	التقدير العام
التقدير	مقبول	جيد جداً	جيد//	جيد جداً	جيد//	جيد//	جيد//	ممتاز	جيد//
الدرجة	٦٤	٧٩	٦٩	٨٢	٧٣	٦٧	٦٦	٨٥	٥٨٥

وقد أعطيت له هذه الشهادة بناء على طلبه وتحت مسئوليته لتقديمها إلى من يهمه الأمر

تحريراً في: ٢٠١٨/١٠/٣٠م

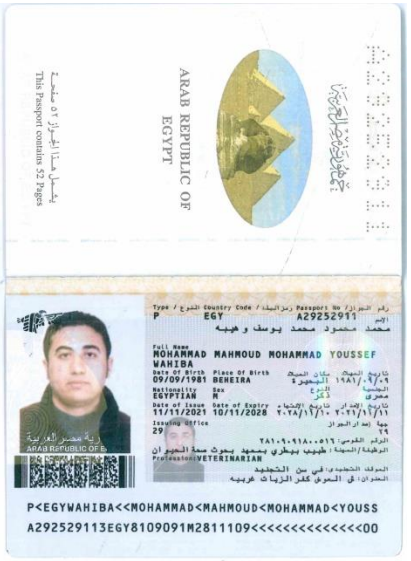
الموظف المختص: أسماء

محمل الطلبة: [illegible]

وكيل الكلية لشئون التعليم والطلاب

عميد الكلية

((أ.د/ محمود عبد الله [illegible])) ((أ.د/ إسماعيل إسماعيل إبراهيم))



يحتوي هذا الجواز ٥٢ صفحة
This Passport contains 52 Pages

ARAB REPUBLIC OF EGYPT

جمهورية مصر العربية

Type / النوع P | Country Code / رمز البلد EGY | Passport No / رقم الجواز A02925911

الإسم محمد محمود محمد يوسف وهيبه

Full Name MOHAMMAD MAHMOUD MOHAMMAD YOUSSEF WAHIBA

Date of Birth 09/09/1981 تاريخ الميلاد ٠٩/٠٩/١٩٨١

Place Of Birth BEHEIRA مكان الميلاد البحيرة

Nationality EGYPTIAN الجنسية مصري

Sex M النوع ذكر

Date of Issue 11/11/2021 تاريخ الإصدار ١١/١١/٢٠٢١

Date of Expiry 10/11/2028 تاريخ الإنتهاء ١٠/١١/٢٠٢٨

Issuing Office جهة إصدار الجواز 29

الرقم القومي: ٢٨١٠٩٠٩١٤٠٠٩١٦

الوظيفة/المهنة: طبيب بيطري

Profession: VETERINARIAN

العنوان: في المنوفية ق: كفر الزيات عربيه

الموقف التجنيد: في سن التجنيد

ARAB REPUBLIC OF EGYPT

P<EGYWAHIBA<<MOHAMMAD<MAHMOUD<MOHAMMAD<YOUSS

A02925911 3EGY8109091M2811109<<<<<<<<<<<<<<00

تتكون كل فقرة من جسم الفقرة وهو القسم الأمامي منها ومن بروزات عظمية عديدة ندعوها النواتئ ترتكز العضلات عليها، ويوجد في مركز كل فقرة حفرة محمية بالعظم المحيط بها، وتشكل الحفر مع بعضها قناة ضمن العمود الفقري هي القناة الشوكية التي يسكنها النخاع الشوكي.

يوجد بين كل فقرتين وسادة اسفنجية اسطوانية الشكل ندعوها القرص بين الفقرات (أو الديسك)، ويتكون هذا القرص من نواة جيلاتينية طرية ندعوها النواة اللبية تتوضع في مركز القرص، ويحيط بها حلقة ليفية قوية مكونة من شرائح متوضعة بشكل متحد المركز تماماً مثل شرائح البصل، ويعتبر القرص بين الفقرات مفصلاً يسمح بحركة العمود الفقري. وهناك ٢٣ قرصاً بين الفقرات يشكلون ثلث ارتفاع العمود الفقري ويخضعون لعمل مستمر، فهذه الأقراص هي التي تمتص الصدمات وتغيرات الضغط التي يخضع لها العمود الفقري، وتكون وظيفة الفقرات الصلبة تحويل هذه الضغوط إلى الأقراص بين الفقرات الملزمة بالتكيف مع الضغوط المختلفة، وفي الحقيقة يؤدي تعرض القرص إلى ضغط شديد إلى تسطح النواة اللبية بغية توزيع الضغط على أكبر مساحة ممكنة، وهذا يؤدي إلى تمدد الحلقة الليفية. وأثناء الراحة يحدث العكس حيث تشتد ألياف الحلقة الليفية ويستعيد القرص ارتفاعه الطبيعي.

بيانات ومعلومات طالب الزواج

بيانات شخصية :

- الإسم/ محمدمحمودمحمديوسف وهيبه
- العمل /مساعدباحث بمعهد بحوث الصحة الحيوانية
- المؤهل / ماجستير البكتيريا والمناعة والفطريات
- (عام 2021/5/19)
- العمر / أربعون عاما
- الحالة الإجتماعية / أعزب لم يسبق لى الإرتباط والزواج
- الخدمة العسكرية / تمت تأديتها
- الإقامة / شارع مجمع المدارس/البيت الأبيض المقابل لمحل ماكس جيم لكمال الأجسام/الدور الثانى ملكى/منزل الأستاذ محمودوهيبه /منطقة العوض/قرية الدلجمون /مركز كفرالزيات / محافظة الغربية
- رقم الموبايل / 01210973074
- البريد الالكتروني : m0hammed_yousef@yahoo.com
- الطول 180سم
- الوزن 100كيلوجرام
- لون البشرة أبيض
- الراتب حوالى 5000 جنيه شهريا
- المطلوب عروسة وزوجة مناسبة لى

بيانات وظيفية :

التعيين / بمعهد بحوث الصحة الحيوانية بالدقي / مصر/فرع طنطا كالتالي :-

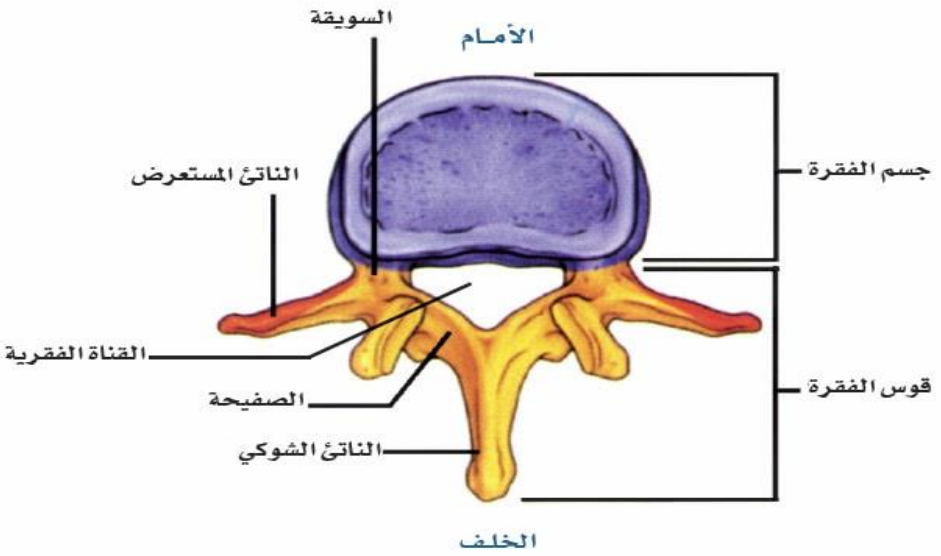
1- طبيب بيطري (من عام 2009إلي 2020)
2- مساعد باحث (من عام 2020/6 الي الوقت الجاري)
3- الحصول على درجة الماجستيرفى البكتيريا والمناعة والفطريات (بتاريخ 2021/5/19)
4- طبيب بيطرى فى الخدمة العسكرية الإلزامية فى الرقابة الصحية على الأغذية

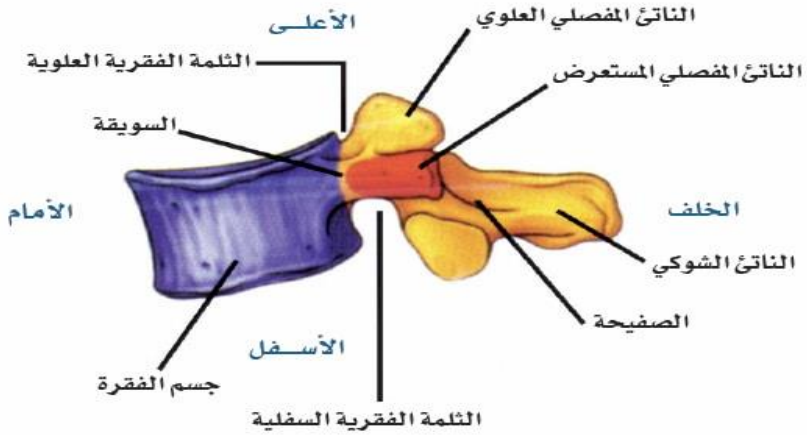
(اللحوم-الألبان ومنتجاتها)

المطلوب من معاليكم زوجة مناسبة

ولسيادتكم وافر التحية والتقديروالإحترام

متمنيا لسعادتكم مزيدا من التقدم والرقي





داء الفقار

دَاء الفَقَار أو تَنَكُّس الفَقار هو انتكاس العمود الفقري لأي سبب. بالمعنى الدقيق، يشير إلى هشاشة العظام في العمود الفقري، وهو تآكل وتمزق العمود الفقري المرتبط بتقدم العمر، وهو السبب الأكثر شيوعًا للإصابة بداء الفقار. تؤثر العملية التنكسية في هشاشة العظام بشكل رئيسي على الأجسام الفقرية، والثقب العصبي، ومفاصل الوجه (متلازمة الوجه). إذا كانت شديدة، فقد تسبب ضغطًا على النخاع الشوكي أو جذور الأعصاب مع اضطرابات حسية أو حركية لاحقة، مثل الألم والتنمل وعدم التوازن وضعف العضلات في الأطراف

عندما تقل المسافة بين فقرتين متجاورتين، قد يؤدي ضغط جذر العصب الخارج من الحبل الشوكي إلى اعتلال الجذور (اضطرابات حسية وحركية، مثل ألم شديد في الرقبة أو الكتف أو الذراع أو الظهر أو الساق، مصحوبًا بضعف العضلات).

أقل شيوعًا، قد يؤدي الضغط المباشر على النخاع الشوكي (عادةً في العمود الفقري العنقي) إلى اعتلال النخاع، الذي يتميز بضعف عالمي، وخلل في المشي، وفقدان التوازن، وفقدان السيطرة على الأمعاء أو المثانة. قد يعاني المريض من صدمات (تنمل) في اليدين والرجلين بسبب انضغاط العصب وقلة تدفق الدم. في حالة إصابة فقرات العنق، يتم تسميتها بداء الفقار الرقبية. يسمى داء الفقار أسفل الظهر داء الفقار القطني. المصطلح مشتق من اليونانية القديمة σπόνδυλος *spóndylos*، «فقرات - »، في الجمع «فقرة»، العمود الفقري».

جمهورية مصر العربية

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية **الطـــب البيـــطري**

أن / **محمـــد محمـــود محمـــد يوسف**

المولود فى **البحيرة** بتاريخ **١٩٨١م**

قد حصل على درجة **البكالوريوس فى العلوم الطبية البيطرية**

فى دور **مايو** عام **٢٠٠٣م** بتقدير **جيـــد جـداً**

مجموع تراكمى **٢٤٧٥ / ٣٣٠٠** النسبة المئوية **٧٥,٠٠%**

و قد اعتمد مجلس الكلية نتيجة إمتحانه فى **٢٠٠٣/٧/١٥م**

ومجلس الجامعة فى **٢٩ /٢٠٠٣/٧م** **بمقتضى قرار مجلس جامعة طنطا**

الموظف المسئول | المراجع | مدير شئون الطلاب | عميد الكلية

تحريراً فى **٢٠١٢/٥/٢٢م**

جمهورية مصر العربية

وزارة الداخلية

قطاع مصلحة الأحوال المدنية

www.cso.gov.eg

صورة قيد الميلاد

رقم القومى ٣٨١٠٩٠٩ ١٨ ٠٥١٦ ٠

بيانات المولود

محمد

الجنسيه :مصر

الديانه :مسلم

النوع :ذكر

محل الميلاد :البحيرة / مركز ابوحمص

تاريخ الميلاد :تسعة من سبتمبر

عام الف و تسعمائه و واحد و ثمانون

بيانات الأب

محمود محمد يوسف وهيبه

الديانه :مسلم

الجنسيه :مصر

بيانات الأم

ليلى محمد عبد المنعم

الديانه :مسلمة

الجنسيه :مصر

م . صحة : ابوحمص المختار رقم القيد :١٧٨٣

س .مدني : مركز ابوحمص ت .القيد :٣٠ /١٠ /١٩٨١

س . امد آر : سجل كفر الزيات ت .امد آر :٢٨ /٠٩ /٢٠١٧

رقم مسلسل ٢١٩٠٤٧٨٥٤٠

توقيع

تأكد من وجود طابع الأمومة والطفولة هلة ، جنيه والعلامة المائية ، نسر شعار الجمهورية ، وشهادة أحوال مدنية.

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((نموذج رقم ٢٥ س))

٣٥٤٣٣٧٣

إصدار ثانى

وزارة الدفاع
شهادة تأدية الخدمة العسكرية
بالقوات المسلحة بجمهورية مصر العربية

تشهد وزارة الدفاع

أن

رقم عسكرى: ٢٠٠٤٥٧٨٨٠١٩٦٦

درجة: ملازم مجند — اسم: محمد محمود محمد يوسف

الرقم القومى: ٢٨١٠٩٠٩١٨٠٠٥١٦ — سجل مدنى: مغر الزيات

محافظة: الغربية

قد انتهت مدة خدمته العسكرية
قوات الجيش
عن مدة خدمة حسنة قدرها

يوم ٥ شهر ١ سنة

بتاريخ يوم ٠١ شهر ٠٩ سنة ٢٠٠٥

بسبب نقله الى الاحتياط تنقضى مدة خدمته بالقوات الاحتياطية

يوم شهر سنة

وكانت درجة أخلاقه أثناء الخدمة قدوة حسنة

تحريرا بتاريخ ٢٠١٩/٠١/٢١

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

بيان مدة الخدمة العسكرية/ كود التعبئة ٥٩٠١٠١٠٠٣

البيان	يوم	شهر	سنة
تاريخ الالتحاق	٢٦	٠٧	٢٠٠٤
تاريخ انهاء الخدمة	٠١	٠٩	٢٠٠٥
اجمالي مدة الخدمة	٥	١	١
المدة الفائدة	٠	٠	٠
صافي مدة الخدمة	٥	١	١

من			إلى			المدة		
يوم	شهر	سنة	يوم	شهر	سنة	يوم	شهر	سنة

الموقف من الخدمة بالقوات الاحتياطية

يتم الرجوع لإدارة شئون ضباط القوات المسلحة لحين بلوغ سن التقاعد للرتبة

تعليمات ونصائح

١ - حافظ على هذه الشهادة سليمة وقدمها إلى كل جهة تطلبها منك.

٢ - الخدمة بالقوات الاحتياطية جزء من الخدمة العسكرية المفروضة عليك قانونا ويجب تأديتها كاملة.

٣ - عندما تستدعى للخدمة بالقوات الاحتياطية عليك التقدم إلى الجهة أو الوحدة التى ستحددها لك تعليمات الاستعداد فى التاريخ الذى يعين لذلك.

٤ - التخلف عن الحضور يوقعك تحت طائلة العقوبة التى قررها القانون لذلك.

٥ - اذا ما حدث ما يسمح لك المغادرة من الخدمة الاحتياطية تقدم إلى إدارة التجنيد والتعبئة لاتخاذ اللازم.

٦ - اذا فقدت منك هذه الشهادة سارع بالاتصال بأجهزة السجلات لاستخراج بدلا منها.

٧ - لا تضع نفسك تحت طائلة القانون سواء بالتعاقد أو العمل بالجيوش الأجنبية بالصفة المدنية أو العسكرية دون الحصول على تصديق مسبق من الجهات المختصة.

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

إدارة السجلات العسكرية - خدمة بمكاتب سجلات الغربية

نقابة الأطباء البيطريين
Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف
المهنة : طبيب بيطرى
رقم القيد : ٣١٦٧٩ / البحيرة
رقم الترخيص : ٣١٧٧٠
سارى حتى : ٢٠٢٠/٠١
الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام
د.حميدة الشيخ

اتحاد نقابات المهن الطبية
نقابة الأطباء البيطريين
www.egy-vet-synd.org

جامعة بنهـــا
مركز الشهادات الموحدة
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026

منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتـــاريخ : ١٩٨١/٩/٩
الجنســـية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكليـة : ٢٧/ ٤/ ٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥/ ٢٠٢١

الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥

المختص ماجد | رئيس القسم أ/أحمد البلاز | وكيل الكلية للدراسات العليا أ.د/ أمانى عبد الرحمن عباس | عميد الكلية

مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

(نموذج رقم ٦٠ ش ش)

جمهورية مصر العربية
وزارة الدفاع
إدارة شئون الضباط للقوات المسلحة
القيد : ٤٩٥٧
التاريخ: ٢٠٠٧/١٢/١٨ م

شهادة
خبرة لضابط إحتياط / مكلف

الى / م.أول احتياط غير مستدعى محمد محمود محمد يوسف

بناء على طلبكم المؤرخ فى ٢٠٠٧٠٣٠٠٤

بشأن إخطاركم بالأعمال التى مارستموها خلال فترة أستدعائكم بالقوات المسلحة

نفيدكم بأنكم قمتم بالأعمال الآتية:

الرقابة الصحية على الأغذية(اللحوم - الألبان ومنتجاتها) .

وذلك خلال الفترة من (٢٠٠٥/٠٩/٠١) الى (٢٠٠٦/١٢/٣١)

و القوات المسلحة تشكركم على خدمتكم و تتمنى لكم التوفيق .

لواء أ.ح / أحمد محمد أحمد نصر
مدير إدارة شئون الضباط للقوات المسلحة
بالتفويض التوقيع (.....)
عقيد / محمد ثروت حسان
رئيس فرع الشئون الشخصيه والخدمه الاجتماعيه

المضاعفات

المضاعفات نادرة ولكن الشديدة في هذا المرض هي قصور العمود الفقري.[١] يحدث هذا بسبب انسداد الشريان الفقري خلال مروره في الثقبة المستعرضة. تصبح مفاصل العمود الفقري صلبة في داء الفقار الرقبية. وهكذا تصبح الخلايا الغضروفية التي تحافظ على القرص محرومة من التغذية وتموت. قد تسبب النبتات العظمية الثانوية تضيقًا للأعصاب الشوكية، يظهر في شكل اعتلال الجذور.

الأسباب

ينتج داء الفقار عن سنوات من الضغط غير الطبيعي المستمر، الناتج عن خلع جزئي للمفصل، أو الإجهاد الناجم عن الرياضة، أو الصدمات الحادة أو المتكررة، أو الوضع السيئ، الذي يتم وضعه على الفقرات والديسك الموجودة بينها.

يتسبب الإجهاد غير الطبيعي في تكوين الجسم لعظام جديدة للتعويض عن توزيع الوزن الجديد. سيؤدي هذا الوزن غير الطبيعي الناتج عن إزاحة العظام إلى حدوث داء الفقار. يمكن أن تؤدي المواقف السيئة وفقدان الانحناءات الطبيعية للعمود الفقري إلى الإصابة بداء الفقار أيضًا. يمكن أن يصيب داء الفقار أي شخص في أي عمر؛ ومع ذلك، فإن كبار السن هم أكثر عرضة للإصابة.

التشخيص

تعد فحوصات التصوير بالرنين المغناطيسي والتصوير المقطعي المحوسب مفيدة في تشخيص الألم ولكنها ليست نهائية بشكل عام ويجب أخذها في الاعتبار مع الفحوصات البدنية والتاريخ.

العلاج

عادة ما يكون العلاج متحفظًا بطبيعته. تثقيف المريض حول تعديلات نمط الحياة، والعقاقير المضادة للالتهابات (NSAIDs) والعلاج الطبيعي، ورعاية تقويم العظام هي أشكال شائعة ، من الرعاية اليدوية التي تساعد في إدارة مثل هذه الحالات.[٥] قد تكون العلاجات البديلة الأخرى مثل التدليك وعلاج نقطة الاثروالبوجا والوخز بالإبر ذات فائدة محدودة. يتم إجراء الجراحة من حين لآخر

تتم الدعوة إلى الجراحة لعلاج اعتلال الجذور العنقي في المرضى الذين يعانون من آلام مستعصية أو أعراض تقدمية أو ضعف لا يتحسن مع العلاج المحافظ.

لا تزال المؤشرات الجراحية لداء الفقار العنقي (CSM) مع اعتلال النخاع مثيرة للجدل إلى حد ما ، ولكن «يوصي معظم الأطباء بالعلاج الجراحي بدلاً من العلاج المحافظ للاعتلال النخاعي المتوسط إلى الشديد»

قد يكون العلاج الطبيعي فعالاً لاستعادة نطاق الحركة والمرونة وتقوية القلب

ومع ذلك، فإن العلاج الطبيعي وعلاج العظام لا يمكن أن «يعالج» الانحطاط، ويرى بعض الناس أن الامتثال القوي لتعديل الوضعية ضروري لتحقيق أقصى فائدة من تخفيف الضغط والتعديلات وإعادة تأهيل المرونة.

ومع ذلك، فقد قيل أن سبب داء الفقار هو ببساطة الشيخوخة (على الرغم من أنه يمكن أن يكون موجودًا في أي عمر)، وغالبًا ما يمارس علاج تعديل الوضع من قبل أولئك الذين لديهم مصلحة مالية (مثل تعويض العمال)[٧] في إثبات أنه ناتج عن ظروف العمل والعادات الجسدية السيئة. فهم علم التشريح هو مفتاح الإدارة المحافظة لداء الفقار.

الجراحة

تهدف الإجراءات الجراحية الحالية المستخدمة في علاج داء الفقار إلى تخفيف علامات وأعراض المرض عن طريق تقليل الضغط في القناة الشوكية (جراحة تخفيف الضغط) و / أو عن طريق التحكم في حركة العمود الفقري (جراحة الدمج).

جراحة تخفيف الضغط: يمكن إجراء جراحة العمود الفقري من كلا المقاربة الأمامية والخلفية. يختلف النهج باختلاف الموقع وسبب ضغط الجذر. بشكل عام، تتم إزالة النباتات العظمية وأجزاء من القرص الفقري.

جراحة الدمج : تُجرى عندما يكون هناك دليل على عدم استقرار العمود الفقري أو سوء المحاذاة. يختلف استخدام الأجهزة (مثل البراغي السنية) في جراحات الاندماج عبر الدراسات.

مساعدباحث /محمدمحمودمحمديوسف وهيبه

اللهم إرزقنا الهجرة والزواج والذرية الموت
والدفن فى بقاع الحرمين الشريفين بسلطانك
ثم عبادك الصالحين
وبارك اللهم فى أعمالنا
وإكتب فيهم الإستفادة اللهم أمين

وسائل الإتصال /٠٠٢٠١٢١٠٩٧٣٠٧٤

E_MAIL : Godlimits@gmail.com

M0hammed_yousef@yahoo.com

Genome2000@yahoo.com

مرض الكبد الدهنى غير الكحولى

التشخيص :

لأن مرض الكبد الدهني غير الكحولي لا يُسبِّب أي أعراض في معظم الحالات، فإنه كثيرًا ما يتمُّ الانتباه إليه طبيًّا عندما تُشير اختبارات تُجرَى لأسباب أخرى إلى مشكلة في الكبد. ويُمكن أن يحدث ذلك إذا كانت حالة الكبد تبدو غير طبيعية عند الفحص بالموجات فوق الصوتية أو إذا كانت نتائج اختبار إنزيم الكبد لديكَ غير طبيعية.

وتشمل الاختبارات التي تُجرَى لمعرفة التشخيص وتحديد مدى شدة المرض ما يلي:

إختبارات الدم

الإجراءات التصويرية

فحص نسيج الكبد

إختبارات الدم:-

تعداد الدم الكامل
اختبارات إنزيمات الكبد ووظائف الكبد
اختبارات التهاب الكبد الفيروسي المزمن (التهاب الكبد A، التهاب الكبد C وغيرهما)
اختبار فحص الداء البطني
سكر الدم الصائم
الهيموغلوبين السكري A1C، والذي يوضح مدى ثبات سكر الدم لديك
فحص الدهون، والذي يقيس دهون الدم، مثل الكوليسترول والدهون الثلاثية

الإجراءات التصويرية:-

تشمل الإجراءات التصويرية المُستخدمة في تشخيص مرض الكبد الدهني غير الكحولي ما يلي:

- تصوير البطن بالموجات فوق الصوتية (الألتراساوند)،

- وهو الاختبار الأول الذي يُجرى عادةً في حالة الاشتباه في الإصابة بأمراض الكبد.

•فحص التصوير المقطعي المُحوسب أو التصوير بالرنين المغناطيسي للبطن.

• تفتقر هذه التقنيات القدرة على تمييز التهاب الكبد الدهني الكحولي من مرض الكبد الدهني غير الكحولي، ومع ذلك قد تظل تُستخدم.

•تصوير المرونة العابر، وهو نمط مُعزز من الموجات فوق الصوتية التي تقيس درجة تيبُّس الكبد لديك. يُشير تيبُّس الكبد لديك إلى وجود تليُّف أو تندُّب.

•تصوير المرونة بالرنين المغناطيسي، يعمل من خلال الدمج بين التصوير بالرنين المغناطيسي والموجات الصوتية لتكوين خريطة مرئية (مُخطط المرونة) تُظهر درجة تيبُّس أنسجة الجسم

فحص نسيج الكبد:-

إذا كانت الفحوصات الأخرى غير قاطعة، فقد يُوصي طبيبك بإجراء لأخذ عينة نسيج من الكبد (خزعة الكبد). تُفحص عينة النسيج في المختبر بحثًا عن علامات الالتهاب والتندُّب.

قد يكون إجراء خزعة الكبد غير مريح، وينطوي على خطورة منخفضة سيُراجعها الطبيب معك بالتفصيل. يُنفذ هذا الإجراء عن طريق إدخال إبرة عبر جدار البطن إلى الكبد.

العلاج:-

بداية العلاج عادةً هي فقدان الوزن من خلال الجمع بين اتِّباع نظام غذائي صحيٍّ، وممارسة التمارين الرياضية. يعالج فقدان الوزن الأسباب التي تُسهِم في الإصابة بمرض الكبد الدهني غير الكحولي. ومن الناحية المثالية، يكون فقدان ١٠% من وزن الجسم أمرًا مرغوبًا فيه، ولكن التحسن في عوامل الخطر يمكن أن يتضح إذا فقدْتَ من ٣ إلى ٥% من وزنك الأولي. وتمثل جراحة إنقاص الوزن أحد الخيارات أيضًا لأولئك الذين يحتاجون لفقد الكثير من الوزن.

قد تكون زراعة الكبد خيارًا لأولئك الذين لديهم تشمع بالكبد؛ نتيجة الإصابة بالتهاب الكبد الدهني غير الكحولي.

العلاجات المحتمَلة في المستقبل :-

لايوجد أي علاج بعقاقير معتمدة من إدارة الغذاء والدواء الأمريكية لمرض الكبد الدهني غير الكحولي، ولكن أُجريت دراسات على بعض الأدوية وكانت ذات نتائج واعدة.

بمساعدة طبيبكَ، يُمكنكَ اتخاذ خطوات لإحكام السيطرة على مرض الكبد الدهني غير الكحولي. يُمكنك:

إنقاص الوزن. في حالِ كنتَ مصابًا بزيادة الوزن أو السمنة، فقُمْ بتقليل عدد السعرات الحرارية التي تتناولها كل يوم، وقُمْ بزيادة نشاطكَ البدني من أجل إنقاص وزنك. تخفيض السعرات الحرارية هو مفتاح إنقاص الوزن والتحكُّم في هذا المرض. إن حاولتَ إنقاص الوزن في السابق، ولكنكَ لم تنجحْ، فاطلبِ المساعدة من طبيبك.

اخترْ نظامًا غذائيًّا صحيًّا. اتبعْ نظامًا غذائيًّا صحيًّا غنيًّا بالفواكه والخضراوات والحبوب الكاملة، وراقِبْ جميع السعرات الحرارية التي تتناولها.

مارِسِ الرياضة وكن أكثر نشاطًا. حاولْ تخصيص ٣٠ دقيقة على الأقل لممارسة التمرينات الرياضية معظم أيام الأسبوع.

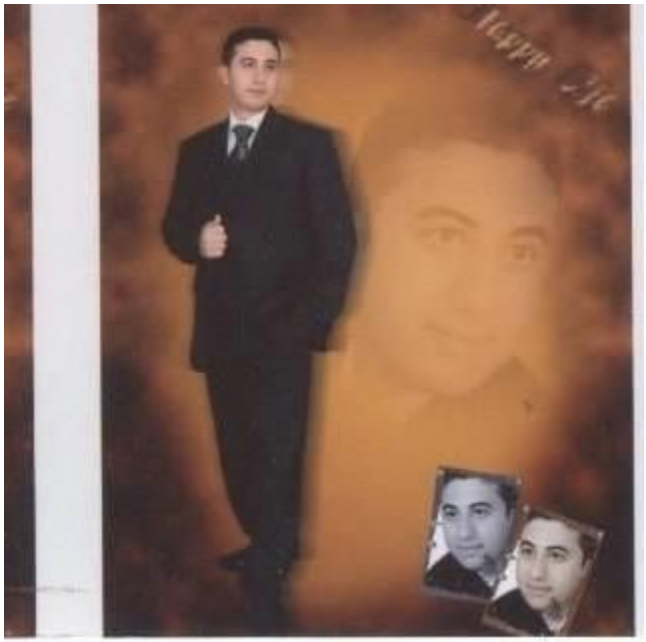
إذا كنتَ بصدد محاولة إنقاص وزنكَ، فستجد أن قيامكَ بالمزيد من التمارين أمر مفيد ونافع. ولكن إذا لم تمارسْ فعلًا التمارين بانتظام، فاحصُلْ على موافقة طبيبكَ أولًا وابدأْ ببطء.

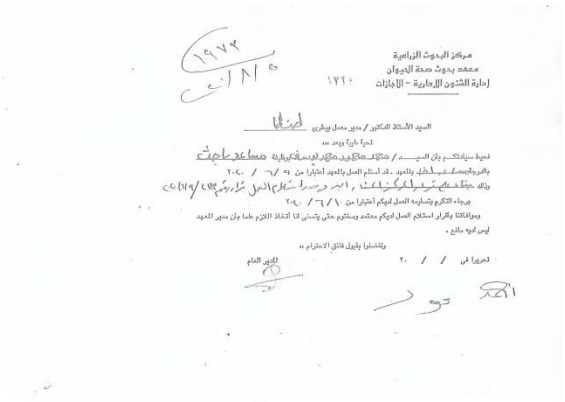
سيطِرْ على مرض السكري. اتَّبِعْ تعليمات طبيبكَ لتبقى مسيطرًا على مرض السكري الخاص بك. تناوَلِ الأدوية الموصوفة لكَ حسب الإرشادات وراقِبْ عن قرب سكر الدم لديك؟

قُمْ بتقليل الكوليسترول. إن اتباع نظام غذائي صحي يعتمد على النباتات وممارسة الرياضة، وتناول الأدوية يُمكن أن يُساعد في الحفاظ على الكوليسترول والدهون الثلاثية عند مستويات صحية.

حماية كبدك. تجنَّبِ الأشياء التي من شأنها أن تضع مزيدًا من الضغوط على الكبد. فعلى سبيل المثال، لا تتناوَل المشروبات الكحولية. اتَّبِعِ التعليمات الخاصة بالأدوية الموصوفةَ لكَ وأيضًا الأدوية المتاحة دون وصفة طبية. تشاوَرْ مع طبيبكَ قبل استخدام أي علاجات عشبية؛ لأنه ليس كل المنتجات العشبية آمنة.

فرط أوكسالات البول والداء الأوكسالى





جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة
بتاريخ : ٩/٩/ ١٩٨١
الجنسية : مصرى
الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٧/ ٤ /٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥ /٢٠٢١
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥
المختص
رئيس القسم
وكيل الكلية للدراسات العليا
عميد الكلية
ا/أحمد البار
أ.د/ أماني عبد الرحمن عباس
مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

نظرة عامة

يحدث فرط أوكسالات البول عندما يحتوي البول على قدر كبير من الأوكسالات. وتُعد الأوكسالات مادة كيميائية طبيعية في الجسم، وتتوفر أيضًا في أنواع طعام معينة. ولكن قد يسبب وجود قدر كبير من الأوكسالات في البول مشاكل خطيرة.

وقد ينتج فرط أوكسالات البول عن اضطراب وراثي (جيني)، أو عن مرض معوي، أو تناول الكثير من الطعام الغني بمادة الأوكسالات.

وتعتمد صحة الكلى طويلة الأمد على التشخيص المبكر لفرط أوكسالات البول وعلاجها العاجل.

يحدث داء الأوكسالي بعد الإصابة بفشل الكلى للأشخاص الذين لديهم أسباب أولية، ومعوية للإصابة بفرط أوكسالات البول، وتراكم الأوكسالات المتزايدة في الدم. مما يؤدي إلى وجود رواسب للأُوكْسالات منها في الأوعية الدموية، وعظام الجسم وأعضائة.

الأعراض

غالبًا ما يكون أول أعراض فرط أوكسالات البول تَكوُّن حصوات الكلى. وقد تشمل أعراض حصوات الكلى ما يلي:

ألم حاد أو مفاجئ في الظهر

ألم مستمر لا يتوقف في المنطقة الواقعة أسفل الضلوع في الظهر (الخاصرة)

ظهور دم في البول

حاجة ملحة إلى التبول المتكرر

ألم مصاحب للتبول

القشعريرة أو حمى

الأسباب

تحدث حالة فرط أوكسالات البول عندما يكون هناك كمية كبيرة من مادة تسمى الأوكسالات في البول. يوجد عدة أنواع من حالة فرط أوكسالات البول:

فرط أوكسالات البول الأولي. فرط أوكسالات البول الأولي يعتبر حالة نادرة وراثية (جينية) تتواجد منذ الولادة. في هذا النوع، لا يُصنّع الكبد ما يكفي من بروتين معين (إنزيم) يمنع الإفراط في إنتاج الأوكسالات، أو أن الإنزيم لا يعمل بشكل صحيح.

يتم التخلص من الأوكسالات الزائدة من خلال كليتيك، في البول. الأوكسالات الإضافية يمكن أن تتحد مع الكالسيوم لتكوين حصوات وبلورات الكلى، والتي يمكن أن تتلف الكلى وتتسبب في توقفها عن العمل (الفشل الكلوي).

تتشكل حصى الكلى مبكرًا وغالبًا ما تسبب أعراضًا خلال الطفولة أو المراهقة. بسبب الكميات الكبيرة المنتجة من الأوكسالات، تفشل الكليتان بحلول مطلع فترة البلوغ وحتي أواسطها لدى العديد من الأشخاص المصابين بفرط أوكسالات البول الأولي. ولكن الفشل الكلوي يمكن أن يحدث في مرحلة مبكرة، مثل مرحلة الرضاعة، في حين أن الآخرين ممن يعانون فرط أوكسالات البول الأولي لا يتطور أبدًا الأمر لديهم إلى الفشل الكلوي. حتى الآن، حدد الخبراء ثلاثة أسباب وراثية مختلفة لفرط أوكسالات البول الأولي.

الداء الأوكسالي. يحدث الداء الأوكسالي إذا كنت مصابًا بفرط أوكسالات البول الأولي وكليتاك تتدهوران. ولأن جسمك لم يعد قادرًا على التخلص من الأوكسالات الإضافية، فإنها تبدأ في التراكم — أولاً في الدم، ثم في عينيك والعظام والجلد والعضلات والأوعية الدموية والقلب وغيرهم من الأعضاء. هذا يمكن أن يسبب مشاكل متعددة.

فرط أوكسالات البول المعوي. العديد من الأمراض المعوية، بما في ذلك مرض كرون ومتلازمة الأمعاء القصيرة الناجمة عن الإجراءات الجراحية، تزيد من امتصاص الأوكسالات من الأطعمة، والذي يمكن أن يزيد من كمية الأوكسالات التي تُفرز في البول.

فرط أوكسالات البول المتعلق بتناول الأطعمة عالية الأوكسالات. تناول كميات كبيرة من الأطعمة التي تحتوي على مستويات عالية من الأوكسالات يمكن أن يزيد من خطر إصابتك بفرط أوكسالات البول أو حصوات الكلى. اطلب من طبيبك أو أخصائي التغذية قائمة بالأطعمة عالية الأوكسالات. تجَنُب الأطعمة عالية الأوكسالات مهمًا للغاية إذا كنت مصابًا بفرط أوكسالات البول المعوي.

المضاعفات

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١/ م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس فى العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جدا) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية الطب البيطري

أن / محمد محمود محمد يوسف

المولود فى البحيرة بتاريخ ١٩٨١م

قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية

فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً

مجموع تراكمى ٢٤٧٥/٣٣٠٠ النسبة المئوية ٧٥,٠٠%

وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م

ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا

الموظف المسئول مستاء المراجع مدير شئون الطلاب عميد الكلية

تحريراً فى ٢٠١٢/٥/٢٢م

نقابة الأطباء البيطريين

Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف

المهنة : طبيب بيطرى

رقم القيد : ٣١٦٧٩ / البحيرة

رقم الترخيص : ٣١٧٧٠

ساري حتى : ٢٠٢٠/٠١

الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام

د.حميدة الشيخ

يمكن أن يؤدي إهمال علاج فرط أوكسالات البول الأولي في النهاية إلى تلف الكلى. وقد تتوقف الكلى عن العمل مع مرور الوقت. وفي بعض الأفراد، تكون هذه العلامة الأولى على الإصابة بالمرض.

وتتضمن علامات وأعراض فشل الكلى ما يلي:

انخفاض إخراج البول أو انعدامه كليًا

الشعور بالمرض أو التعب العام

فقدان الشهية والغثيان والقيء

شحوب لون البشرة المرتبط بفقر الدم

تورم اليدين والقدمين

يمكن أن يؤدي الداء الأوكسالي إلى مضاعفات متنوعة خارج الكلى، والتي تتضمن أمراض العظم وفقر الدم وقُرح الجلد ومشكلات القلب والعين، وفي الأطفال، فشل النمو الطبيعي.

التشخيص :-

ويقوم الطبيب بإجراء فحص طبي شامل والذي يتضمن مناقشة حول التاريخ الطبي والنظام الغذائي. تشمل فحوصات تشخيص فرط أوكسالات البول ما يلي

فحوصات البول، لقياس الأوكسالات ومستويات نواتج الأيض الأخرى في البول

فحوصات ألم، لبيان وظيفة الكلى ومستويات الأوكسالات في الدم

تحليل الحصوات، لتحديد مكونات حصوات الكلى التي تخرج مع البول أو بالإزالة الجراحية

الأشعة السينية على الكلى أو الموجات فوق الصوتية أو الفحص بالتصوير المقطعي المحوسب، بحثًا عن وجود أيّ حصوات في الكلى أو ترسبات أوكسالات الكالسيوم

وبعد الفحص الأول، قد ينصح الطبيب بالمزيد من الفحوصات لتأكيد التشخيص ومعرفة ما إذا كان المرض أصاب أجزاء أخرى من الجسم. قد تشمل تلك الاختبارات:

فحص الحمض النووي بحثًا عن الأسباب الوراثية (فرط أوكسالات البول الأولي)

خزعة الكلى بحثًا عن ترسبات الأوكسلات

مخطط صدى القلب لفحص ترسبات الأوكسلات في القلب

فحص العين لفحص ترسبات الأوكسالات في العين

خزعة نخاع العظم لفحص ترسبات الأوكسلات في العظام

خزعة الكبد لمعرفة حالات نقص الإنزيمات، حيث تلزم فقط في حالات نادرة عندما لا تكشف الفحوصات الوراثية عن سبب فرط أوكسالات البول

في حالة تشخيص إصابة المريض بفرط أوكسالات البول الأولي، يكون الأشقاء معرضين لخطر الإصابة بالمرض وينبغي خضوعهم للفحوصات. في حالة إصابة الأبناء بفرط أوكسالات البول الأولي، يمكن مراعاة إجراء الفحوصات الوراثية في حالة التخطيط لإنجاب مزيد من الأطفال. ويمكن للاستشاريين الوراثيين الطبيين المتمرسين أن يساعدوا في تقديم التوجيه بشأن القرارات والفحوصات.

العلاج:-

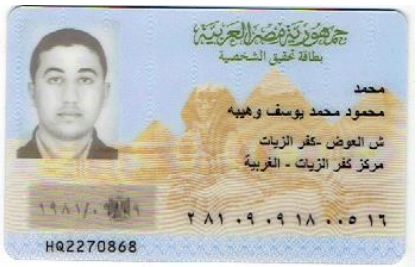
يعتمد العلاج على نوع مرض فرط أوكسالات البول وأعراضه وحدته ومدى جودة استجابتك للعلاج.

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

يمكن أن تكون جرعات فيتامين بى ستة فعالة في تقليل الأوكسالات في البول لدى بعض الأشخاص المصابين بفرط أوكسالات البول الأول.

تساعد مستحضرات السترات الفموية على منع تكوين بلورات أوكسالات الكالسيوم.

تناوُل كميات كبيرة من السوائل. إذا كانت الكليتان لا تزالان تعملان بصورة طبيعية، فمن المحتمل أن ينصحك طبيبك بشرب المزيد من الماء أو غيره من السوائل. إذ يعمل ذلك على غسيل الكلى ويمنع تراكم بلورات الأوكسالات ويساعد على منع تكوُّن حصوات الكلى.



التغيرات في النظام الغذائي. بوجه عام، تزداد أهمية العناية بنظامك الغذائي إذا كنت تعاني من فرط أوكسالات البول المعوي أو الغذائي. قد يوصي طبيبك بإدخال تغييرات إلى نظامك الغذائي، مثل تقليل الأطعمة التي تحتوي على نسبة عالية من الأوكسالات والحد من تناوُل الملح وتقليل البروتين الحيواني والسكر (شراب الذرة عالي الفركتوز). ويمكن أن تساعد مثل هذه التغييرات الغذائية على خفض مستويات الأوكسالات في البول. لكن قد لا تكون القيود الغذائية فعالة لدى جميع الأشخاص المصابين بفرط أوكسالات البول الأولي. اتبع توصيات طبيبك.

الأطعمة التي تحتوي على كميات كبيرة من الأكسالات

السبانخ.

اللوز.

الشمندر واللفت.

التوت.

زبدة الجوز.

المكسرات والكاجو.

الشوكولاتة والكاكاو.

ما أسباب تكون أوكسالات الكالسيوم؟

عدم الحصول على كمية كافية من السوائل. الإفراط في تناول نظام غذائي غني بالأوكسالات، أو البروتين، أو الملح. فرط نشاط جارات الغدة الدرقية. مرض التهاب الأمعاء.

السيطرة على حصوات الكلى

تنتشر حالات حصوات الكلى لدى المصابين بفرط أوكسالات البول ولكن لا تحتاج إلى العلاج دائمًا. إذا كانت الحصوات الكبيرة بالكلى تسبب الألم أو تعوق تدفق البول، فقد يحتاج المريض إلى إزالتها أو تفتيتها لتخرج مع البول.

الغسيل والزراعة

وفقًا لدرجة خطورة حالة فرط أوكسالات البول التي تعاني منها، ربما تصاب في نهاية المطاف بفقدان وظائف الكلى. ربما يكون غسيل الكلى مفيدًا بصورة مؤقتة، ولكنه لا يتوافق مع كمية الأوكسالات التي يتم إنتاجها. ربما تؤدي زراعة الكلى أو زراعة الكلى والكبد إلى التعافي من أنواع وراثية معينة من فرط أوكسالات البول (فرط أوكسالات البول من النوع الأول).

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE
DEPARTMENT OF BACTERIOLOGY, IMMUNOLOGY, AND MYCOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Youssef Wahhabi

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

Pure microbiology

Branches of microbiology

Bacteriology: the study of bacteria.
Mycology: the study of fungi.
Protozoology: the study of protozoa.
Phycology/algology: the study of algae.
Parasitology: the study of parasites.
Immunology: the study of the immune system.
Virology: the study of viruses.
Nematology: the study of nematodes.

https://en.wikipedia.org/wiki/Branches_of_microbiology

GENERAL

BACTERIOLOGY, IMMUNOLOGY, MYCOLOGY

INTRODUCTION TO MICROBIOLOGY

MICROBIOLOGY

IS DEFINED AS

(THE STUDY OF ORGANISMS TOO SMALL TO BE CLEARLY SEEN BY THE NAKED EYES).

THESE ORGANISMS INCLUDE VIRUSES, BACTERIA, ALGEA, FUNGI, ARCHAEA AND PROTOZOA. WHEN STUDYING MICROORGANISMS , MICROBIOLOGISTS ARE CONCERNED WITH CHARACTERISTICS AND FUNCTIONS SUCH AS MORPHOLOGY ,CYTOLOGY, PHYSIOLOGY , ECOLOGY , TAXONOMY , GENETICS , AND MOLECULAR BIOLOGY .

TAXONOMY التصنيف الحيوى ECOLOGY علم البيئة

Leeuwenhoek
(Live 1632 -1723)
is the first person to use the microscopes to observe microbes: he made small handheld microscopes: he called ((microorganisms as animalcules)).

خلايا بدائيات النوى خلية بدائية النواة Procaryotes

TAXONOMY AND CLASSIFICATION OF PROCARYOTES

HEAKEL (1866) WAS THE FIREST TO CREATE ANATURAL KINGDOM FOR MICROORGANISMS , HE PLACED ALL UNICELLULAR (MICROSCOPIC) IN ANEW KINGDOM CALLED PROTISTA , SEPERATED FROM PLANTS (PLANTAE) AND ANIMALS (ANIMALIA) , WHICH WERE MULTICELLULAR (MACROSCOPIC) ORGANISMS .

PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS
PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS
ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

IN (1950) : THE DEVELOPMENT OF ELECTRON MICROSCOPE

THE ELECTRON MICROSCOPE MORE ONE MILLION POWERFUL THAN THE LIGHT MICROSCOPE.

SO BY THE ELECTRON MICROSCOPE THEY FIND ANOTHER KINGDOM CALLED MONERA (MONERES) : INCLUDES MICROSCOPIC UNICELLULAR ORGANISMS BUT NOT HAVE THE NUCLEAR MEMBRANE .

PROTISTA :
REMAINED AS AKINGDOM OF UNICELLULAR EUCARYOTIC MICROORGANISMS .

WHITTAKER : REFINED THE ECOSYSTEM INTO FIVE KINGDOMS IN (1967) BY IDENTIFYING THE FUNGI AS ASEPERATE MULTICELLULAR EUOCARYOTIC KINGDOM OF ORGANISMS , DISTINGUISHED BY THEIR ABSORPTIVE MODE OF HETEROTROPHIC MODE OF NUTRITION .

يتكون النظام البيئى إلى خمس مملكات حيوية :

THE FIVE KINGDOMS OF THE ECOSYSTEM :

1-PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS

2-PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS

3-ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

4- MONERA : UNICELLULAR MICRORGANISMS NOT HAVE NUCLEAR MEMBRANE.

5- FUNGI : MULTICELLULAR OF EUCRAYOTIC ORGANISMS ITS LENGHTH 4 MICROMETERS IN LENGTH .

BACTERIA IS 1 MICROMETERS IN LENGTH

FUNGI IS 4 MICROMETERS IN LENGTH

FIVE KINGDOMS OF THE ECOSYSTEM AS FELLOW :

ANIMALIA :INCLUDES VERTEBRATES AND ARTHROPODES

PLANTEA : INCLUDES GREEN ALGEA

FUNGI : INCLUDES MOLDS AND YEASTS

MONERA : INCLUDES ARCHAEA ,BACTERIA,MYCOPLASMA

PROTESTA : INCLUDES RED ALGEA , AMOEBAS, WATER MOLDS .

What are 5 characteristics of fungi?

Characteristics of Fungi

Fungi are eukaryotic, non-vascular, non-motile and heterotrophic organisms.

They may be unicellular or filamentous.

They reproduce by means of spores.

They Have Two Types Of Reproduction Sexually And Asexually Reproduction .

Fungi exhibit the phenomenon of alternation of generation.

Fungi lack chlorophyll and hence cannot perform photosynthesis.

BACTERIAL MORPHOLOGY

PLEASE CHANGES THE PICTURES TO BE ELECTRONIC MICROSOPIC RECOGNITIONS

PROKARYOTIC CELLS ARE GENERALLY MUCH SMALLER THAN EUKARYOTIC CELLS

BACTERIA REMARKKABLY VARY IN SIZES , SHAPES AND ARRANGEMENT

MOST OF BACTERIA COME IN BASIC FOUR SHAPES :

1-COCCI 2- ROD OR BACILLI 3-ARCH

4-SPIRAL

1-COCCUS

THE COCCI ARE SPHERICAL OR OVAL BACTERIA HAVING ONE OF SEVERAL DISTICNT ARRANGEMENTS :

1-DIPLOCOCCUS :PAIR OF COCCI

2-STREPTOCOCCUS : CHAIN OF SEPERATE COCCI

3-TETRAD : SQUARE OF FOUR COOCI

4-SARCINA :CUBE OF 8 COCCI

5-STAPHYLOCOCCUS:COCCI IN IRREGULAR , OFTEN GRAP LIKE CLUSTE

2-BACILLUS :

BACILLI ARE ROD SHAPED BACTERIA , THEY DEVIDED IN ONE PLANE PRODUCING ONE OF THE FOLLOWING :

1-BACILLIUS : ASINGLE BACILLUS (CYLINDRICAL)

2- COCCO BACILLUS HAVE ABULGING MOSTLY IN THE MIDDLE

3-STREPTO BACILLI : ACHAIN OF SEPERATED BACILLUS TO BE FACILITATING CONGUGATION

4-SPIRAL : AHELICAL OR CORKSCREW –SHAPED BACTERIUM .SPIRALS COME IN 1 OF 3 FORMS :

لولبى

1-VIBRIO : CURVED OR COMA SHAPED ROD

2-SPIRILLUM : THICH RIGID SPIRAL

3-SPIROCHETE : THIN FLEXIBLE SPIRAL

PROCARYOTIC CELL STRUCTURES AND FUNCTIONS

THE WORD PROCARYOTE WILL BE USED IN AGENERAL SENSE TO INCLUDE BOTH BACTERIAE AND ARCHAEA
(MYCOPLASMA IN ASEPERATE TAXONOMY)
ARCHAE ARE MICROORGANISMS THEY ARE NO MORE RELATED TO BACTERIA THAN TO EUKARYOTES . THEY SHARE SOME TRAITS WITH BACTERIA ,SUCH AS HAVING ASINGLE CIRCULAR MOLECULE OF DNA ,THE PRESCENSE OF MORE MOBILE PIECES OF GENATIC MATERIALES CALLED PLASMIDS ,SIMILAR ENZYMES FOR PRODUCING COPIES OF DNA . HOWEVER THEIR METHODS OF PROTIEN PRODUCTION AND ORGANIZATION OF THEIR GENATIC MATERIAL BEARS MORE SIMILARITY TO EUBACTERIAL CELLS .
ARCHAE INCLUDES MORE ARELATIVELY SMALL NUMBERS OF MICROORGANISMS THEY ARE INHABITING ENVIRONMENTS WHICH ARE TOO HARSH FOR OTHER MICROBES.SUCH AS ENVIRONMENTS INCLUDES HOT ,MOLTEN VENTS AS AT THE BOTTOM OF OCEANS , AND THE HIGHLY SALT WATER OF THE GREAT SALT LAKE AND THE DEAD SEA.

البحر الميت – البحيرة المالحة الأمريكية العظيمة ولاية أوتاها كلاهما مالحان

فتحات مصهورة --- لقدذكرت من علامات الساعة

الكبرى جفاف بحيرة طبريا وهى من أعذب وأحلى مياه العالمين

البحر المميت الأردن قوم لوط العرب

البحيرة المالحة الأمريكية بولاية أوتاها ليس لها مسمى فاحش

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الشكوى الإدارية فى ملفات الرقابة الإدارية المصرية

DIFFERENCES BETWEEN PROKARYOTE AND EUKARYOTE AND ARCHAEA :

THE COMPARISONS WILL COVER THIS POWER POINTS :

1-CELL CONFIGURATIONS 2-NUCLAR MEMBRANE 3-NUMBER OF CHROMOSOME 4-CHROMOSOME TOPOLOGY 5-MURIEN IN CELL WALL 6-CELL MEMBRANE LIPIDS 7- CELL MEMBRANE STEROLS 8- ORGANELLES ESPECIALLY MITOCHONDRIA AND CHLOROPLASTS) 9-RIBOSOMES SIZES 10- CYTOPLASMIC STREAMING 11-MEIOSIS AND MITOSIS) 12- TRANSCRIPTIONS AND TRANSLATIONS COUPLED 13- AMINO ACID INITIATING PROTEIN SYNTHESIS

14- PROTEIN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHENICOL

15- PROTEIN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN

DIFFERENCES BETWEEN EUKARYOTE, PROKARYOTE,ARCHAEA			
PROPERITY	BIOLOGICAL DOMAIN		
	EUCARYA	BACTERIA	ARCHAEA
CELL CONFIGURATION	EUCARYOTIC	PROCARYOTIC	PROCARYOTIC
NUCLEAR MEMBRANE	PRESENT	ABSENT	ABSENT
NUMBER OF CHROMOSOMES	MORE THAN ONE	ONE	ONE
CHROMOSOME TOPOLOGY	LINEAR	CIRCULAR	CIRCULAR
MUREIN IN CELL WALL	-	+	-
CELL MEMBRANE LIPIDS	ESTER-LINKED GLYCERIDES UNBRANCHED POLYUNSATURATED	ESTER-LINKED GLYCERIDES: UNBRANCHED SATURATED OR MONOUNSATURATED	ETHER –LINKED BRANCHED: SATURATED
CELL MEMBRANE STEROLS	PRESENT	ABSENT	ABSENT

ORGANELLES (MITOCHONDERIA AND CHLOROPLASTS)	PRESENT	ABSENT	ABSENT
RIBOSOME SIZE	80S	70S	70S
CYTOPLASMIC STREAMING	+	–	–
MEIOSIS AND MITOSIS	PRESENT	ABSENT	ABSENT
TRANSCRIPITION AND TRANSLATION COUPLED	–	+	+
AMINOACID INITIATING PROTIEN SYNTHESIS	METHIONINE	N-FORMYLE METHIONINE	METHIONINE
PROTIEN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHINCOL	–	+	–
PROTIEN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN	+	–	+

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

BACTERIAL STRUCTURES

INCLUDES :

1- ESSENTIAL BACTERIAL STRUCTURES

2- NON ESSENTIAL BACTERIAL STRUCTURS

1- ESSENTIAL BACTERIAL STRUCTURES

INCLUDES :

1-CELL WALL AND

2-PROTOPLASM

2-PROTOPLASM INCLUDES :

A-CYTOPLASM AND

B-CELL MAMBRANE

A-CYTOPLASM INCLUDES :

CHROMOSOMES ,MESOSOMES,RIBOSOMES, INCLUSIONS BODIES.

2-NON ESSENTIAL BACTERIAL STRUCTURES :

INCLUDES :

CAPSULES,FLAGELLAE,PILI,
SPORES,
PLASMIDES.

1-ESSENTIAL BACTERIAL STRUCTURES :

1-CELL WALL :

1-ONE OF THE MOST IMPORTANT PARTS OF THE PROKAYOTIC CELL.

2-MOST OF PROCAYOTES HAVE ARIGID CELL WALL.

3-THE CELL WALL IS CELL MADE OF PEPTIDOGLYCANS: POLYMER OF PEPTIDES AND GLYCANS.

PEPTIDES (TYPICAL 4 AMINOACIDES LONG ,CROSS-LINKED TO OTHER CHAINS)

GLYCANS (MADE OF ALTERNATING AMINO SUGARS)

4- PEPTIDOGLYCAN IS AHUGE POLYMER OF INTERLOCKING CHAINS OF IDENTICAL MONOMERS.

5- THE BACKBONE OF PEPTIDOGLYCAN MOLECULES COMPOSED OF TWO DERIVATIVES OF GLUCOSE :
N-ACETYLE GLUCOSAMINE(NAG)
AND N-ACETYLMURAMIC ACID (NAM)
THE (NAG) AND (NAM) STRANDS ARE CONNECTED BY INTERPEPTIDE BRIDGES

6-FUNCTIONS OF CELL WALL :

1-SUPPORTS THE WEAK CYTOPLASMIC MEMBRANE AGAINST INTERNAL OSMOTIC PRESSURE .

2-RESPONSIBLE FOR THE SHAPE OF BACTERIAE AND THEIR GRAM STAIN REACTIONS .

3-INTEGRITY OF THE CELL WALL ESSENTIAL FOR VIABILITY OF BACTERIAE .

4-PLAY ROLE IN CELLS DIVISIONS AND ANTIGENICITY OF THE BACTERIAE .

IN THE GRAM POSITIVE BACTERIAE THOSE RETAIN CRYSTAL VIOLET DYE WHEN SUBJECTED TO THE GRAM STAINING PROCEDURES , THE CELL WALL CONSISTS OF SEVERAL LAYERS OF PEPTIDO GLYCAN . TEICHOIC ACIDS RUNING PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.
TEICHOIC ACIDS IS AGROUP OF MOLECULES WHICH ARE UNIQUE TO GRAM POSITIVE CELL WALL .

STRUCTURE OF THE GRAM POSITIVE BACTERIAL CELL WALL .THE CELL WALL IS RELATIVELY THICK AND CONSISTS OF MANY LAYERS OF PEPTIDO GLYCANS INTERSPERSED WITH TECHOIC ACIDS THAT RUN PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.

IN THE GRAM NEGATIVE BACTERIAE (WHICH DO NOT RETAIN CRYSTAL VIOLET DYE EXCEPT ALITTLE) :

THE CELL WALL COMPOSED OF ASINGLE LAYER OF PEPTIDO GLYCANS SURROUNDED BY AMEMBRANOUS STRUCTURE CALLED THE OUTERMEMBRANE.

THE OUTER MEMBRANE OF GRAM NEGATIVE BACTERIAE INVARIABLY CONTAINS AUNIQUE COMPONENT,LIPOPOLYSACCHARIDES (LPS OR ENDOTOXIN), WHICH IS TOXIC IN ANIMALS.

STRUCTURE OF THE GRAM NEGATIVE- BACTERIAE CELL WALL.

THE WALL IS RELATIVELY THIN AND CONTAINS MUCH LESS PEPTIDO GLYCANS THAN THE GRAM POSITIVE BACTERIAL WALL. ALSO,TEICHOIC ACIDS ARE ABSENT. HOWEVER, THE GRAM NEGATIVE-CELL WALL CONSISTS OF AN OUTERMEMBRANE THAT IS OUTSIDES OF THE PEPTIDO GLYCAN LAYER.THE OUTERMEMBRANE IS ATTACHED TO THE PEPTIDO GLYCAN SHEET BY AUNIQUE GROUP OF LIPOPROTIEN MOLECULES.

THE PROFILES OF THE CELL ENVELOPE OF THE GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE.

THE GRAM POSITIVE WALL IS AUNIFORMLY THICK LAYER EXTERNAL TO THE PLASMA MEMBRANE . IT IS COMPOSED MAINLY OF PEPTIDOGLYCAN (MEURIN).

THE GRAM NEGATIVE WALL

APPEARS THIN AND MULTILAYERED.

IT IS CONSIST OF ARELATIVELY THIN PEPTIDOGLYCAN SHEET BETWEEN THE PLASMA MEMBRANE AND APHOSPHOLIPID-LIPOPOLYSACCHARIDE OUTERMEMBRANE.

THE SPACE BETWEEN THE INNER PLASMA MEMBRANE AND OUTER MEMBRANE WHERE IN THE PEPTIDOGLCAN RESIDES IS CALLED PERIPLASM.

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

1-BACTERIAL MEMBRANES COMPOSED OF 40% TO 60% PROTIEN.BACTERIAL PLASMA MEMEBRANES USUALY HAVE AHIGHER PROPORTION OF PROTIEN THAN DO EUCARYOTIC MEMBRANES,PERSUMABLY BECOUSE THEY FULFIL SO MANY DIFFERENT FUNCTIONS THAT ARE CARRIED OUT BY OTHER ORGANELLE MEMBRANES IN EUCARYOTES.

FUNCTIONS OF-Be- CELL MEMBRANE:

1-OSMOTIC BARRIER FOR MAINTAINING DIFFERENCE IN SOLUTE CONTENT BETWEEN CYTOPLASM AND EXTERNAL ENVIRONMENT.

2-PERMIT PASSIVE DIFFUSION INWARD FROM OUTWARD OF WATER AND LIPID SOULABLE MATERIAL.

3-SELECTIVE TRANSPORT OF SPECIFIC NUTRIENT SOLUTE IN TO THE CELL.

4-ENZYMATIC ACITIVITY AS IT CONTAINS PERMEASE,RESPIRATORY,CYTOCHROME AND POLYMERZING ENZYMES.

5-ENERGY GENERATING FUNCTIONS,INVOLVING RESPIRATORY AND PHOTOSYNTHETIC ELECTRON TRANSPORT SYSTEMS,ESTABLISHMENT OF PROTON MOTIVE FORCE, AND TRANSMEMBRANOUS, ATP-SYNTHESIZING ATPase.

6-COORDINATIONS OF DNA REPLICATIONS AND SEGREGATION WITH SEPTUM FORMATION AND CELL DIVISION.

2-THE PERIPLASM :

IS ASPACE BETWEEN PLASMA MEMBRANE AND CELL WALL.

IT IS VISIBLE IN GRAM NEGATIVE AND DIFFICULT IN GRAM POSITIVE BACTERIAE.

IT CONTAIN VARIOUS HYDROLYTIC ENZYMES AND BINDING PROTIENS THAT SPECIFICALLY BIND SUGARS ,AMINOACIDS AND INORGANIC IONS ,THESE IONS AND PROTIENS THAT AID IN TRANSPORT VARIOUS COMPOUNDS INTO AND OUT BACTERIAL CYTOPLASM.

3-THE CYTOPLASM :

1-THE CYTOPLASM OF BACTERIAL CELL IS VISCOUS WATERY SOLUTION CONTAIN ORGANIC AND INORGANIC SUBSTANCES AND RIBOSOMES.
2-IT HAS NO MITOCHONDERIA,NO ENDOPLASMIC RETICULUM, NO GOLGI APPARATUS.
3-IT COMPOSED FROM RNA.
4-IT CONTAINS BACTERIAL CHROMOSOMES (NECLEOID),RIBOSOMES,INCLUSION GRANULES AND MESOSOMES.

4-THE BACTERIAL CHROMOSOMES (NECLOID):

IT COMPRISES ACLOSED LOOP OF DOUBLE STRANDED SUPERCOILED DNA.(NO NUCLEAR MEMBRANE).IN ADDITION ,SOME BACTERIAE CONTAIN ADDITIONAL DNA IN THE FORM OF SMALL ,SELF REPLICATING EXTERA-CHROMOSOMAL ELEMINTS CALLED PLASMIDS.THESE DO NOT CARRY ANY GENES ESSENTIAL FOR GROWTH AND REPRODUCTIONS,AND THUS THE CELL MAY SERVIVE WITHOUT THEM. THE PLASMIDS CAN BE VERY IMPORTANT HOWEVER,AS THEY MAY INCLUDE GENES ENCODING TOXINS OR RESISTANCES TO ANTIBIOTICS ,AND CAN BE PASSED FROM CELL TO CELL.

5-RIBOSOMES:

THESE ARE THE SITES OF PROTIENS SYNTHESIS.ALTHOUGH THEY CARRY OUT ASIMILAR FUNCTIONS,THE RIBOSOMES OF PROCARYOTIC CELLS ARE SMALLER AND LIGHTER THAN THIER EUCARYOTIC CELL.
PROCARYOTIC RIBOSOMES ARE 70 S ,WHILE THOSE OF EUCARYOTES ARE 80 S.

6-MESOSOMES:

IT IS ACONVOLUTED BODY INVAGINATED FROM CYTOPLASMIC MEMBRANE INTO CYTOPLASM.
1-ENTER IN THE MECHANISM OF CELL DIVISION.
2-HAS AROLE IN SPORULATION OF BACTERIA.
3- ACT AS AMITOCHONDERIA OF EUCARYOTIC CELL.
4-HAS AROLE IN RESPIRATION OF BACTERIA.
5-EXCRETE MATERIALS FROM CYTOPLASM TO EXTERIOR.

7-INCLUSIONS BODIES :

1-IT WERE ASTORED RESERVES OF NUTRIENTS OF AGGREGATES OF EXCESS SUBSTANCES .

2-IT VARRIES ACCORDINDG TO TYPE OF BACTERIAL STRUCTURE OR GENOMES.

E.G: FOR EXAMPLES:

a-VOLUTIN (METACHROMIC GRANULES) IN PASTEURELLA SPP.

b-LIPIDS GRANULES IN TUBERCLE BACILLI.

NON ESSENTIAL STRUCTURES

1-CAPSULES AND -S LAYERS

2-FELAGELLAE

3-PILI

4-SPORE

5-PLASIMDS .

1-CAPSULES AND S LAYERS :

A-SOME BACTERIA HAVE ALAYER OF MATERIAL LYING OUTSIDE THE CELL WALL

B-WHEN THE LAYER IS WELL RECOGNIZED AND EASILY WASHED OFF IT IS CALLED ATRUE CAPSULE.

C-ATRUE CAPSULE IS A DISCRETE DETECTABLE LAYER OF POLYSACCHARIDES DEPOSITED OUTSIDE THE CELL WALL.

D-ALESS DISCRETE STRUCTURE OR MATRIX WHICH EMBEDS THE CELL CALLED ASLIME LAYER OR BIOFILM.

E-CAPSULES AND SLIME LAYERS USUALLY ARE COMPOSED OF POLYSACCHARIDES .

F-BUT THEY MAY BE CONSTRUCTED FROM OTHER MATERIALS .

G-FOR EXAMPLE BACILLUS ANTHRAX HAVE ACAPSULE OF POLY-GLUTAMIC ACIDS .

H-CAPSULES ARE CLEARLY VISIBLE BY IN LIGHT MICROSCOPE.

I-MANY GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE HAVE AREGULARLY STRUCTURED LAYER AN S LAYER ON THIER SURFACE . THE S-LAYER HAS APATTERN SOMETHING LIKE FLOOR TILES AND IS COMPOSED OF PROTIEN OR GLYCOPROTIEN.

N.B THERE AN ACONFESSION THAT THERE AN ELECTRON MICROSOPE IMPLENTATIONS IN THE UNIVERSITY BOOK.

J-BACTERIAL CAPSULES OUTLINED BY INDIA INK VIEWED BY LIGHT MICROSCOPY .THIS ATRUE CAPSULE ,ADISCRETE LAYER OF POLYSACCHARIDE SURROUNDING THE CELLS.SOMETIMES BACTERIAL CELLS ARE EMBEDDED MORE RANDOMLY IN APOLYSACCHARIDES MATRIX IN ASLIME LAYER OR BIOFILM.

FUNCTIONS OF CAPSULES :

A-PROTECT THE CELL WALL AGAINST ANTIBACTERIAL AGENTS .
B-PROTECT THE BACTERIAL CELLS AGAINST INGESTION BY THE PHAGOCYTIC CELLS .
C-RESPONSIBLE OF VIRULENCE OF THE ORGANISMS
D-THE S -LAYER CAN FUNCTION AS AN ADHESION ,ENABLING THE BACTERIUM TO ADHERE TO HOST CELL MEMEBRANES AND ENVIRONMENTAL SURFACES IN ORDER TO COLONIZES .
E-MANY OF THE CELL –ASSOCIATED PROTIEN ADHESINS USED BY PATHOGENS ARE COMPONENTS OF THE S-LAYER.

2-FLAGELLA :

A-MOST MOTILE BACTERIA MOVE BY USE OF FELAGELLA (S.FLALGELLUM) ,
B-THREADS LIKE LOCOMOTOR APPENDAGES EXTENDING OUTWARD FROM THE PLASMAMEMBRANE.
C-THEY ARE SLENDER , RIGID STRUCTURES ,ABOUT 20 NM ACROSS AND UP TO 15 OR 20 MICRO METER LONG .
D-FLAGELLA ARE SO THIN THEY CAN NOT BE OBSERVED DIRECTLY WITH –A BRIGHT FIELD MICROSCOPE.
E-BUT MUST BE STAINED WITH ESPECIAL TECHNIQUES DESIGNED TO INCREASE THEIR THICKNESS.

F-BACTERIAL SPECIES OFTEN DIFFER DISTINCTIVELY IN THEIR PATTERNS OF FLAGELLAE DISTRIBUTIONS :

1-MONOTRICHOUS BACTERIA (TRICHOUS MEANS HAIR) HAVE ONE FELAGELLUM:IF IT IS LOCATED AT THE END IT IS SAID TO BE POLAR FLAGELLUM

2-AMPHITRICHOUS BACTERIA (AMPHI MEANS ON BOTH SIDES) LE FELAGELLUM ز HAVE ASINGLE FELAGELLUM ON THE BOTH POLE. ON THE BOTH POLE>BOTH POLE BE POLAR FLAGELLUM E PLASMAMEMBRANE MEBRANE LE ,ADISCRETE LAYER OF POLYSACCHARIDE SURR

3-LOPHOTRICHOUS BACTERIA (LOPHO MEANS TUFT) HAVE ACLUSTER OF FELAGELLA AT ONE OR BOTH ENDS .

4-PERITRICHOUS (PERI MEANS AROUND) BACTERIA HAVE FLAGELLA THAT ARE SPREAD FAIRLY EVENLY OVER THE WHOLE SURFACE . FELLAGELATION PATTERNS ARE VERY USEFUL IN IDENTIFYING BACTERIA.

G-FELAGELLAR ULTRASTRUCTURE:

A-THE LONGEST AND MOST OBIVIOUS PORTION IS THE FILAMENT ,WHICH EXTENDEDS FROM THE CELL SURFACE TO THE TIP.

b-abasal body is embedded in the cell :the body has four rings connected to a centeral rod . the outer l and p rings associated with the lipopolysaccharides and peptidoglycan layers ,respectively .

d-ashort curved segment ,the hook ,links the filament to to its basal bodies and acts as aflexible coupling.

THE FILAMENT IS AHOLLOW ,RIGID CYLINDER CONSTERUCTED OF ASINGLE PROTIEN CALLED FLAGELLIN .

3-FIMBRIAE AND PILI :

FIMBRIAE AND PILLI ARE SHORT-HAIR LIKE STRUCTURES ON THE SURFACES OF PROCARYOTIC CELLS .LIKES FELAGELLA ,THEY ARE COMPOSED OF PROTIEN .FIMBRIAE ARE SHORTER AND STIFFER THAN FELAGELLA AND SLIGHTELY SMALLER IN DIAMETER .GENERALLY ,FIMBERIAE HAVE NOTHING TO DO WITH BACTERIAL MOVEMENT (THERE ARE EXCEPTIONS, E.G . TWITCHING, MOVEMENT ON PSEUDOMONAS).

N.B FIMBERIAE ARE MOST COMMON IN GRAM NEGATIVE BACTERIAE , BUT OCCUR IN SOME GRAM POSITIVE BACTERIAE AS WELL.

FIMBRIAE ARE MOST OFTEN INVOLVED IN ADHERENCE OF BACTERIA TO SURFACES ,SUBSTRATES AND OTHER CELLS OR ISSUES IN NATURE. IN E.CLOI , ASPECIALIZED TYPE OF PILUS , THE F OR SEX PILUS, APPARENTELY STABLIZES MATING BACTERIA DURING THE PROCESS OF CONJUGATION , BUT THE FUNTION OF THE SMALLER , MORE NUMEROUS COMMON PILLI ARE QUITE DIFFERENT .

COMMON PILI (ALMOST ALWAYS CALLED FIMBRIAE) ARE USUALLY INVOLVED IN SPECIFIC AHERENCE (ATTATCHMENT) OF PROCARYOTES TO SURFACES IN NATURE.

IN THE MEDICAL SITUATIONS , THEY ARE MAJOR DETERMINANTS OF BACTERIAL VIRULENCE BECOUSE THEY ALLOW PATHOGENS TO ATTACH TO (COLONIZE) TISSUES AND OR TO RESIST ATTACK BY PHAGOCYTIC WHITE BLOOD CELLS . FOR EXAMPLE , PATHOGENIC NEISSERIA GONORHOEAE ADHERES SPECIFICALLY TO THE HUMAN CERVICAL OR URETHERAL EPITHELIUM BY MEANS OF FIMBERIAE : ENTEROTOXIGENIC STRAINS OF ESCHERICHIA COLI ADHERE TO THE MUCOSAL EPITHELIUM OF THE INTESTINE BY MEANS OF SPECIFIC FIMBRIAE .

PROPERTY	FLAGELLA	PILI
COMPOSITION	FLAGELLIN, APROTIEN	PILIN, APROTIEN
OCCUERENCE	MANY BACTERIAL RODS,BUT FEW COCCI	MANY GRAM NEGATICE AND COCCI
FUNCTION	MOTION	CONJUGATION,ATTACHMENT
SHAPE	LONG AND THIN	SHORT AND STIFF
ORIGIN	HOOK LIKE INSERTION TO BASAL GRANULE INSIDE CELL WALL	CYTOPLASM

4- THE BACTERIAL END SPORES:

1-ANUMBER OF GRAM POSITIVE BACTERIA CAN FORM ASPECIAL RESISTANT , DORMANT STRUCTURE CALLED AN END SPORES .
2-END SPORES DEVELOP WITHIN VEGETATIVE BACTERIAL CELLS OF SEVERAL GENERA
3-SEVERAL GENERA INCLUDES : 1- BACILLUS LIKES ANTHRAX AND CLOSTRIDIUM (RODS) AND 2- COCCI LIKES SPOROSARCINA
3- AND OTHERS .

4- THESE STRUCTURES ARE EXTRA ORDINARILY RESISTANT TO INVIRONMENTAL STRESSES SUCH AS HEAT,ULTRA VIOLET, RADIATION,GAMMA RADIATIONS, CHEMICAL DISINFECTANTS,DESICCATIONS,

5- IN FACTS ,SOME ENDO SPORES HAVE REMAINED VIABLES FOR AROUNDING 100,000 YEARS.

6-BECOUSES OF THEIR RESISTANSE AND INFACT THAT SEVERAL SPECIES OF ENDOSPORES –FORMING BACTERIA ARE DANGEROUS PATHOGENS , ENDOSOPRES ARE OF GREAT PRACTICAL IMPORTANCE IN FOOD,INDUSTERIAL ,AND MEDICAL MICROBILOGY ,

7-THIS BECOUSE ENDOSPORES OFTEN RESIST SURVIVE BOILING FOR AN HOUR OR MORE.

8-SPORES POSITIONS IN THE MOTHER CELLS , (SPORANGIUM) FREQUENTLY DIFFERS AMONG SPECIES ,MAKING IT OF CONSIDERABLE VALUE IN INDENTIFICATIONS , AS IT MAY LOCATE CENTRAL, TERMINAL OR SUBTERMINAL .

9- SPORE STRUCTURES :

A-THE SPORES OFTEN IS SURROUNDED BY ATHIN DELICATE COVERING CALLED THE EXOSPORIUM .

B-ASPORE COAT LIES BENEATH THE EXOSPORIUM ,IS COMPOSED OF SEVERAL PROTIENS LAYERS AND MAY BE FAIRLY THICK, IT IS IMPERMEABLE AND RESPONSIBLE FOR SPORES RESISTANCES TO CHEMICALS .

C-THE CORTEX, WHICH MAY OCCUPY AS MUCH AS HALF THE SPORE VOLUME , RESISTS BENEATH THE SPORE COAT IT WERE MADE OF PEPTIDO GLYCANS .

D-THE SPORE CELL WALL (OR CORE WALL) IS INSIDES THE CORTEX AND SERROUNDS THE PROTOPLASTS OR CORES .

E- THE CORES HAS THE NORMAL CELL STRUCTURES SUCH AS RIBOSOMES AND ANEUCLOIDES , BUT IT METABOLICALLY INACTIVE.

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :بكر

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :

1- 15 % OF THE SPORES DRY WEIGHT CONSISTS OF DIPICOLINC ACIDS COMPLEXED WITH CALICIUM IONS WHICH IS LOCATED IN THE CORES. IT HAS LONG BEEN THOUGHT THAT DIPICOLINIC ACIDS WAS DIRECTLY INVOLVED IN SPORES HEAT RESISTANCES .

2- DEHYDERATIONS OF THE PROTOPLASTS VERY IMPORTANT IN HEAT RESISTANCES .

3- THE CORTEX MAY OSMOTICALLY REMOVE WATER FROM PROTOPLASTS ,THERBY PROTECTING IT FROM HEAT AND RADIATIONS DAMAGES.

4-THE SPORES COATS ALSO SEEMS TO PROTECTING AGAINST ENZYMES AND CHEMICALS SUCH AS HYDROGEN PEROXIDES .

11- SPORE FORMATIONS (SPOROGENESIS OR SPORULATIONS):

1-NORMALLY COMMENCES WHEN GROWTH CEASES DUE TO LACK OF NUTRIENTS . IT IS ACOMPLEX PROCESS AND MAY DEVIDED INTO SEVEN STAGES :

1-AN AXIAL FILAMENT OF NUCLEAR MATERIAL FORMS .

2-INWARD FOLDING OF THE CELL MEMBRANE TO ENCLOSE PART OF THE DNA ORODUCE THE FORESPORE SEPTUM .

3-THE MEMBRANE CONTINUES TO GROW TO ENGULFS THE IMMATURE SPORE IN THE SECOND MEMBRANE.

4-CORTEX IS LAID DOWN IN THE SPACE BETWEEN THE TWO MEMBRANES ,AND BOTH CALCIUM AND DIPICOLINIC ACIDS ARE ACCUMULATED .

5-PROTIEN COATS THEN ARE FORMED AROUND THE CORTEX .

6-MATURATIONS OF THE SPORE OCCURS.

7-LYTING ENZYMES DESTROY THE SPORANGIUM RELEASING THE SPORE.

SPORULATION REQIURES ONLY ABOUT TEN HOURS IN BACILLUS MEGATERIUM .

BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE
DEPARTMENT OF CLINICAL PATHOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Youssef Wahhabi

جامعة بنها
كلية الطب البيطرى
قسم الباثولوجيا الإكلينكية
مقدمة فى الباثولوجيا الإكلينكية الحديثة
تحت تحرير وإشراف أ.د خالد محمد رئيس القسم
كتبها ونسقها مساعدباحث/
محمدمحمودمحمديوسف وهيبه
ماجستير البكتيريا والمناعة والفطريات
معهدبحوث الصحة الحيوانية
خاص بمحمدوهيبه

BODY FLUIDS EXAMINATION

FIRST EDITION
COURSE NO. 178

EDITED BY
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PREFACE

THIS IS THE FIREST EDITION OF TUMOR MARKERS. IT IS ATEACHING BOOK WHICH IS DIRECTED AS APOST GRADUATE COURSE. IT IS ORGANIZED BY CLINICAL PATHOLOGY DEPARTMENT FOR POST GRADUATED STUDENT S IN CLINICAL PATHOLOGY OR OTHER ESPACIALISTS.

I ACKNOWLEDGE AND THANK ALL STAFFS IN CLINICAL PATHOLOGY DEPARTMENT FOR THEIR EFFORT AND I HOPE VERY MUCH THAT, IN THIS EDITION, I HAVE MANAGED TO MAINTAIN THE STANDERED OF CONTENT, PRESENTATION AND ACCESSIBILITY.

IN-DEPTH COVERAGE OF THE MORE ESTABLISHED SUBJECTS OF TUMOR MARKERS BOOK INCLUDING : THE CELLULAR AND LIQUID CONSTITUENTS CHARACTERISTICS OF BLOOD , CHARACTERISTICS FEATURES OF NORMAL AND ABNORMAL BLOOD CELL FORMATION AND MATURATION ,

THE STAGES OF LEUCKOCYTE DEVELOPMENT, THE ORDER OF MATURATION AND SERIES AND CHARACTERISTICS OF EACH STAGE WITH VARIATION , OCCURRENCE OF LEUCKEMIA AND ITS CLASSIFICATIONS AND TYPES , THE APPROPIATE SIGNS AND SYMPTOMS FOR THE STAGE OR TYPE OF LEUCKEMIA .

ALSO MOST COMMON TUMOR MARKERS AND ELISA TEST USED FOR ITS DETECTION IN BLOOD ARE WELL COVERED. THESE TOPICS HAVE BECOME IMPORTANT IN TEACHING TUMOR AND ITS BIOMARKERS AS APART OF CLINICAL PATHOLOGY. I BELIEVE I HAVE ACHIEVED HIGH LEVEL OF ACCESSIBILITY AND EASY READING.

KHALID MOHAMED

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ELECTROLYTES AND TRACE ELEMENTS

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WATER METABOLISM

1-WATER CONSTITUTES ABOUT 45-70 % OF TOTAL BODY WEIGHT .

2- BODY FLUIDS ARE DIVIDED IN TWO COMPARTMENTS:

(A)- INTRA CELLULAR (ICF), WHICH WHICH CONSTITUTES 65-75 % OF TOTAL BODY WATER AND 35-45 % TOTAL BODY WEIGHT .

(B)- EXTERA CELLULAR FLUIDS (ECF) CONSTITUTES ABOUT 25 % OF TOTAL BODY WATER AND 15 % OF TOTAL BODY WEIGHT .

3-)- EXTERA CELLULAR FLUIDS (ECF) FOUND IN 3 LOCATIONS :

(A)- INTRA VASCULAR (PLASMA)

(B)-INTRASTITIAL (LYMPH)

(C) – TRANS CELLULAR (IT INCLUDES : CSF ,JOINT FLUIDS ,PERITONEAL PERICARDIAL, PLEURAL AND LIQUID IN INTESTINE).

4-DECREASED TOTAL BODY WATER LEADS TO DEHYDERATION

5-DEHYDERATION IS MANIFESTED BY :

(A)-DECRASED BODY WIEGHT

(B)LOSS OF SKIN ELASTICITY

(C)-DRYNESS OF MUCOUS MEMBRANE

(D)-RETRACTIONOF EYES OF THE EYES

6- ELECTROLYTES AND WATER MOVES FREELY BETWEEN THE ICF AND ECF .

MOVEMENT OF WATER IS DEPENDANT UPON ON HYDROSTATIC PRESSURE AND OSMOTIC PRESUURE OF ECF PROTIENS

7- OSMOTIC PRESSURE OF ANY FLUIDS IS DIRECTLY RELATED TO THE NUMBER OF PARTICLES IN A UNIT OF SOLVENT AND IS MEASURED BY OSMOLE OR MILLIOSMOLE .

8- OSMOLE IS THE MOLECULAR WEIGHT OF THE SUBSTANCE MULTIPLIED BY THE NUMBER OF IONS INTO WHICH IT DISSOCIATES .

9- THE NORMAL OSMOLALITY OF ECF IS ABOUT 300 MOSM /KG

10-ECF CONTAINS SODIUM NA , CHLORIDE CL AND BICARBONATE HCO3

11- ICF CONTAINS K POTTASIUM ,MG MAGNESIUM ,ORGANIC PHOSPHATES AND PROTIEN.

12-SERUM OSMOLALITY CAN BE ESTIMATED BY USING THE CONCETRATION OF NA SODIUM , AND K POTTASIUM AS FOLLOW :

MOSM / KG = 2(NA +K) +BUN/2.8+GLUCOSE/18

13-SERUM OSMOLALITY IS USED AS AMEASURE OF ECF OSMOLALITY .

14-HYPER OSMOLALITY IS FOUND IN CASE OF :
(A)-HYPERNATREMIA
(B)-ACCUMLATION OF ENDOGENOUS SOLUTES SUCH AS GLUCOSE ,KETONE BODIES , AND UREA
15- HYPOOSMOLALITY IS FOUND IN CASE OF HYPONATERMIA
16-OSMORECEPTORS SENSE CHANGES IN OSMOLALITY . WITH HYPER OSMOLALITY (HYPONATREMIA) , OSMORECEPTORS STIMULATES VASOPRESSIN OR ADH SECRETION FROM THE PITUITARY GLAND AND STIMULATE THIRST . THIRST IS STIMULATE BY AS ALITTLE AS

1-2 % CHANGES IN OSMOLALITY .THE END RESULTS IS WATER RETENTION BY THE KIDNEY AND INCREASED WATER INTAKE . INCREASES IN FREE WATER WILL THUS WILL REDUCE SODIUM CONCENTERATION ,OPPOSITECHANGES OCCUR WITH HYPOOSMOLALITY.

17-BARORECEPTORS ARE SENSITIVE TO CHANGES IN EFFECTIVE CIRCULATING VOLUME (ECV) THE ECV IS THAT PART OF THE EXTRACELLULAR FLUID THAT IS IN THE ARTERIAL SYSTEM AND IS EFFECTIVELY PERFUSING THE TISSUES . IT USUALLY VARIES DIRECTLY WITH ECF VOLUME .

WITH HYPOVOLEMIA (DECREASED ECV), BAROECEPTORS STIMULATES THE RENIN-ANGIOTENSIN SYSTEM , THE END RESULT BEING MINERALOCORTICOID (ALDOSTERONE) RELEASE FROM THE ADRENAL CORTEX.

ALDOSTERONE INCREASED ABSORPTION OF NACL AND PROMOTES THE EXCRETION OF POTASSIUM AND HYDROGEN IN THE DISTAL TUBULES OF THE NEPHRON , NACL RETENTION PROMOTES WATER REABSORPTION , THIS CORRECTING THE HYPOVOLEMIA . HYPOVOLEMIA ALSO STIMULATES THIRST (A DECREASE IN ECV OF 7-10 % IS REQUIRED FOR STIMULATION). OPPOSITE CHANGES OCCUR WITH HYPER VOLEMIA

ANTIDIURETIC HORMONE

ADH ACTS ON CHIEF CELLS IN COLLECTING TUBULES , INCREASING PERMEABILITY OF THE TUBULES TO WATER AND UREA . ADH ACTS VIA ADENYLE CYCLASE WHICH OPEN CHANNELS IN THE TUBULES ,CALLED AQUA-PORES , FACILITATING WATER ABSORPTION .ADH IS SECRETED BY HYPOTHALAMUS IN RESPONSE TO OSMOTIC (MAINLY DETERMINED BY NA SODIUM) AND NON OSMOTIC (VOLUME DEPLETION ,NAUSIA ,FEAR,ANXIETY,PAIN,EXERCISE AND DRUGS) STIMULI.

SEVERAL THINGS INHIBIT THE ACTION OF ADH ON RENAL TUBULES AND THUS AFFECT CONCENTRATING ABILITY .

THESE INHIBITORS OF ADH INCLUDE :

(1)-HYPOKALEMIA

(2)-HYPERCALCEMIA

(3)- CORTICOSTEROIDS

(4-)-PROSTAGLANDIN E

IN ANY OF THE ABOVE CONDITIONS URINE CONCENTRATING ABILITY IS IMPAIRED FOR REASONS OTHER THAN PHYSIACL LOSS OF FUNCTIONAL NEPHRONS .

(1- SODIUM)

SYNONUM : SERUM SODIUM NA +

SPECIMEN : SERUM OR PLASMA (HEPARINE) I ML .

REFERENCE VALUE : 136-145 MEQ/LITRE ; SI UNITS : 136-145 MMOL/LITRE

METHODS : ION-SELECTIVE ELECTRODE ,SPECTROPHOTOMETER ,FLAMEPHOTOMETER .

DESCRIPTION :

1-SODIUM IS THE MAIN CATION OF EXTERACELLULAR FLUIDS WHERE AHALF OF TOTAL BODY SODIUM IS PRESENT .MOST OF THE REST SODIUM IS PRESENT IN BONES IN STABLE FORM .

2- SODIUM HAS TWO MAIN FUNCTIONS :

A-REGULATION OF ACID BASE BALANCE

B-PLAY AN IMPORTANT ROLE IN REGULATION OF OSMOTIC PRESSURE

3-THE QUANTITY OF SODIUM IS CONTROLLED BY SODIUM INTAKE AND LOSS.

4- HORMONAL CONTROL :

A-IT IS PEFORMED BY ALDOSTERONE HORMONE ,WHICH WHICH SECRETED BY ADRENAL S GLANDS : ITS ACTION IS PERFORMED ON THE RENAL TUBULES WHERE IT CONTROLS EXCRETION AND REABSORPTION OF SODIUM .

B-MOST OF SODIUM PRESENTED TO RENAL TUBULES IS REABSORBED (APPROXIMATELY 90 %) UNDER CONTROL OF ALDOSTERONE HORMONE .

C-IF THERE IS AN EXCESS OF BODY SODIUM ,ALDOSTERONE SECRETION IS REDUCED AND SODIUM IS EXCRETED THROUGH KIDNEYS :BUT IF THE SODIUM IS REDUCED ,ALDOSTERONE SECRETION AND PRODUCTION IS INCREASED AND SODIUM IS ALMOST COMPLETELY REABSORPED .

N.B SODIUM REABSORPTION REQIURES AN EQUIVALENT PASSAGE OF HYDROGEN OR PUTASSIUM IONS IN THE OPPOSITE DIRECTION .

D-EXCRETION OF SODIUM :

IT IS DONE THROUGH :

1-MAINLY KIDNEYS THROUGH URINE.

2-SWEAT .

3-SECRETIONS OF DIGESTIVE TRACTS .

4-THROUGH STOOL.

HYPONATREMIA

DECREASE IN PLASMA SODIUM CONCENTRATION IS CALLED HYPONATERMIA
IT OCCURS MOST FREQUENTLY BECOUSE OF EXCESSIVE SODIUM LOSS THROUGH THE FOLLOWING ROUTS :

1-DIGESTIVE TRACT :

A-DIARRHEA b – VOMITING

2-URINARY TRACT :

A-RENAL DISEASES RENAL FAILURE b– USING OF DIURETICS-

D- DIABETES MELLITUS AND HYPER GLYCEMIA .

3-EXCESSIVE SWEATING WITHOUT ADEQUATE SODIUM INTAKE .

4- EXCESSIVE EXUDATION FROM BURNS OR OPEN WOUNDS WHEN SODIUM INTAKE IS INDEQUATE .

5-ADRENOCORTICAL INSUFFICIENCY (ADDISON S DISEASE).

6-LIPEMIA IN WHICH LIPIDS DISPLACES ASIGNIFICANT VOLUME OF LIQUID PHASE OF PLASMA MAY RESULT IN ALOW PLASMA SODIUM ,SIMPLY BECOUSE SMALLER VOLUME OF LIQUID PHASE .

6-CONGESTIVE HEART FAILURE .

7-ADMINISTERATION OF HYPOTONIC SOLUTION SUCH AS 5% DEXTROSE .

9-METABOLIC ACIDOSIS .

10-INSUFFICIENT INTAKE .

HYPERNATREMIA

1-HYPERNATREMIA IS ARARE CONDITION .

2-IT OCCURS WHEN THERE IS LOSS OF FLUIDS CONTAIN ASMALL AMOUNT OF SODIUM AS IN DIABETES INSPIDUS .

3-EXCESSIVE SODIUM INTAKE WITH LIMITED LIQIUD INTAKE .

4-REDUCED GLOMERULAR FILTERATION RATE AS IN RENAL FAILURE.

5-HYPER ALDOSTERONISM .

2- POTASSIUM

SYNONUM : SERUM K+

SPECIMEN: SERUM OR PLASMA HEPARINE I ML .

REFERENCE VALUE : 3.9 – 5.8 % MEQ/L .

METHODS : ION –SELECTIVE ELERCTRODE, FLAMOPHOTOMETER, SPECTROPHOTOMETER .

DESCRIPTION :

1-POTASSIUM IS THE MAN CATIONS IN THE CELLS OR INTRACELLULAR

2- FUNCTION OF POTASSIUM ARE :

A-CONTROL OF MUSCULAR ACTIVITY

B- CONTROL NEURAL EXCITABILITY

3-EXCRETION OF POTASSIUM IS DONE THROUGH:

A-KIDNEY

B-STOOL

C-SWEAT

D- DIGESTIVE FLUIDS

4-ALDOSTERONE FACILITATES EXCRETION OF POTASSIUM AS IT INCREASE SODIUM REABSORPTION BY PROMOTING EXCHANGE OF SODIUM FOR POTASSIUM .

POTASSIUM IS PRESENT IN MORE HIGH CONCENTERATION INTRACELLULAR ,SO THAT NECROSIS OF TISSUES OR DESTRUCTION OF RBCS CAUSES HIGH SERUM POTASSIUM LEVEL .

HYPO KALEMIA :

1-DIARRHEA AND VOMITTING ESPECIALLY WHEN REPLACEMENT FLUIDS IS POTASSIUM (K+)FREE .
2-IATROGENIC AS IN CASE OF PROLONGED ADMINSTERATION OF MINERALOCORTICOID.
3-HYPERALDOSTERONISM
4-ALKALOSIS ,POTASSIUM DECREASES BECOUSES IT ENTERS INSIDE THE CELLS EXCHANGING WITH HYDROGEN IONS AND ALSO INCREASE EXCRETION THROUGH URINE.
5-BICARBONATE THERAPY
6-DIURETIC TREATMENT
7-RENAL TUBULAR ACIDOSIS
8-PROLONGED ANOREXIA
9-EXCESSIVE SWAETING
10-CHRONIC RENAL FAILURE
11-INSULIN TREATMENT IN KETOACIDOTIC DIABETES (WILL CARRY POTASSIUM INTO THE CELLS ALONG WITH GLUCOSE).

HYPER KALEMIA

1-ACUTE RENAL FAILURE WHERE THERE IS ANURIA OR OLIGOURIA

2-HYPOADRENOCORTICISM (ADDISONS DISEASE)

3-USING OF POTASSIUM SPARCING DIURETICS

4-INCREASE INTAKE (AS POTASSIUM CHLORIDE (KCL) USED IN CONGESTIVE HEART FAILURE)

5-ACIDOSIS

6-DIFFUSE CELL DEATH SECOUNDARY TO SHOCK ,CIRCULATORY STASIS CHEMOTHERAPY

7-FACTITIOUS (ERRORS DURING COLLECTION OF SAMPLE):

a-HEMOLYSIS –b-DELAYED SEPARATION OF CLOTTED BLOOD (DUE TO POTASSIUM LEAKAGE) –c- COLLECTION IN POTASSIUM HEPARINE.

CYSTATIN C

CYSTATIN C

SPECIMEN: SERUM OR PLASMA (HEPARIN)
METHODOLOGY: IMMUNOASSAY
REFERENCE VALUE: 0, 53 - 0, 95 mg /L

Description

Description :

1- CYSTATIN C IS ACYSTIENE PROTIENASE INHIBITOR .

2-ASMALL MOLECULE THAT IS PRODUCED BY NUCLEATED CELLS .

3-SERUM CYSTATIN C IS FILTERED OUT OF BLOOD BY THE KIDNEYS AND THAT SERVES AS AMEASURE OF KIDNEYS FUNCTION.

4- ITS MOLECULAR MASS ALLOWS IT TO BE FREELY FILTERED BY THE GLOMERULAR MEMBRANE IN THE KIDNEYS.

5-SINCE IT IS FORMED AT ACOSTANT RATE AND FREELY FILTERED BY THE HEALTHY KIDNEYS.

6-CYSTATIN C IS AGOOD MARKER OF RENAL FUNCTION.

7-SERUM CONCENTERATIONS OF CYSTATIN C ARE ALMOST TOTALLY DEPENDANT ON GLOMERULAR FILTERATION RATE (GFR) .

8-AREDUCTION IN (GFR) CAUSES ARISE IN THE CONCENTERATION OF CYSTATIN C.

9-CYSTATIN C IS ABETTER MARKER OF THE GLOMERULAR FILTERATION RATE AND HENCE OF KIDNEY FUNCTION THAN CREATININE WHICH WAS THE MOST COMMONLY USED MEASURE OF KIDNEY FUNCTION.

10- THE LEVELS OF CYSTATIN C ARE INDEPENDENT OF WEIGHT AND HEIGHT ,MUSCLE MASS,AGE, (OVER AYEAR OF AGE) ,SEX,AND FACTORS WHICH HAVE BEEN DOMENSTERATED TO AFFECT CREATININE VALUES,IN ADDITION,ARISE IN CREATININE DOES NOT BECOME EVIDENT UNTIL (THE GFR) HAS FALLEN BY APPROXIMATELY 50%.

11- MEASUTEMENTS CAN BE MADE AND INTERPRETED FROM ASINGLE RANDOM SAMPLE.

12- CYSTATIN C MAY IS USED AS AN ALTERNATIVE TO CREATININE AND CREATININE CLEARANCE TO SCREEN FOR AND MONITOR KIDNEY DYSFUNCTION IN THOSE WITH KNOWN OR SUSPECTED KIDNEY DISEASES.

13- IT MAY BE ESPECIALLY USEFUL IN THOSE CASES WHERE CREATININE MEASUREMENT IS NOT APPROPRIATE: FOR INSTANCE IN THOSE WHO HAVE LIVER CIRRHOSIS, ARE VERY OBESE, ARE MALNOURISHED OR HAVE AREDUCED MUSCLE MASS.

14- MEASURING CYSTATIN C MAY ALSO IS USEFUL IN THE EARLY DETECTION OF KIDNEY DISEASE WHEN OTHER PARAMETERS MIGHT STILL BE NORMAL, ESPECIALLY IN THE ELDERLY.

THANK YOU

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THE TOPIC TODAY

Renal failure

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
28109091800516/2004578801966

Renal failure is a state in which the body's metabolism is disturbed as a consequence of dysfunction of the kidneys, characterized by numerous manifestations, which are attributable to the imbalance between the rate of production of various chemical substances by the body and their rates of the excretion by the kidneys.

Kidneys become unable to perform its regulator role of the electrolytes, acid-base balance and excretion of waste products.

(Syndrome of renal insufficiency)

There are two characteristics syndromes of renal failure:-

1-acute renal failure.

2-chronic renal failure.

They Are So Two Distinct , And Each One Consider Alone .

1-Acute Renal Failure :

Definition :

It Means Acute Suppression Of Kidney Filtration Resulting In Rapidly Increasing Azotemia , With Or Without Oliguria .

Acute Renal Failure Can Be Divided Into Two Phases :

1-Oilguric Phase .

2-Diuretic Phase .

1-Oilguric Phase Which Is Characterized By :

A-Sever Decrease Of Urine Volume .

B-Increased Level Of Urea And Creatinine .

C-Decreased Serum Na+ And Hyper Kalemia.

D-Sediments Shows Protein , Red Cells ,Epithelial Cells And Casts.

2-Diuretic Phase :

Begins 2 Weeks Post Onset , Gradual Increase Of Urine Volume Indicating Nephrons Recovery (Continues For 7-10 Days).

Causes Of Acute Renal Failure :-

1-Pre Renal Causes
(Vascular Diseases).
2-Renal Causes .
3-Post Renal Causes .

1-Pre Renal Causes (Vascular Diseases):-

Sudden Vasoconstriction Of Renal Arteries In The Most Important Cause Of Acute Renal Failure.

Vasoconstriction Occurs In The Following Condition:-

A-Sudden Decrease In Blood Volume Due To Hemorrhage.

B-Cardiac Failure:

C-The Presence Of The Large Quantities Of Free HB And Myoglobin.

D-Certain Completion Of Pregnancy As Eclampsia ,Concealed Accidentals Hemorrhage And Abortion.

C-Postoperative.

:++++2-Renal Causes : It Includes

A-Glomular Diseases:

*-Acute Glomerulonephritis Caused By Salmonella ,Shigella And Toxoplasma.

*-Pyelonephritis With Rapidly Necrosis Caused By Coryne Bacterium Renale.

*-Systemic Lupus Erythematosis.

*-Membranous Glomerulonephritis.

B-Vascular Diseases:

*-Acute Bilateral Renal Artery Obstruction As Caused Thrombosis And Or Embolism.

*-Disseminated Intravascular Coagulation (DIC).

*-Thrombotic Thrombocytopenic Purpura (TTP).

C-Interstitial And Tubular Diseases:

*-Acute Interstitial Nephritis Caused By Leptospira.

*-Acute Tubular Necrosis :This May Occur Toxic Or Ischaemic.

3-Post Renal Causes :

***-Acute Urinary Tract Obstruction Occur Due To :**

A-Stones In The Pelvis Or Ureters Or Urethera Or Urinary Bladder.

B-Prostatic Tumor Or Stressful Hypertrophy .

C-Blockage Of The Tubules By Casts Or Crystals Of Uric Acid Or Sulphonamides

Laboratory Findings :

1-Passage Of Small Amount Of Dark Urine (Oliguria).

2- Changes In Blood.

3- Hematological Changes .

1-Passage Of Small Amount Of Dark Urine (Oliguria)

A-Urine Contains Protein , Blood And Cellular Debris .

B-Specific Gravity Is Variable (Usually High)

2- Changes In Blood:

A-Retention And Increased Level Of Urea And Creatinine ,Potassium ,Magnesium ,Phosphate other Anions.

B-Metabolic Acidosis.

C-The Blood Calcium Is Some Lowered Secondarily To The Raised Blood Phosphate.

D-A considerable Increase In Extracellular Water.

E-Decrease In The Serum Concentration Of Sodium And Chloride.

3- Hematological Changes Include :

A-Rapidly Developing Anemia (Normocytic Normochromic Anemia) And Is Accentuated By Diluting Effect Of The Increased Blood Volume Secondarily To Water Retention .

B-Hemolytic Type Of Anemia Occurs To Due To Uremia.

C- Neutrophilic Leuckocytosis Is Also Present.

JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
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THE TOPIC TODAY
CHRONIC RENAL FAILURE

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
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DEFINITION :

IT MEANS CHRONIC INSUFFICIENCY OF RENAL EXCRETORY AND REGULATING FUNCTIONS LEADING TO UREMIA

CHRONIC RENAL FAILURE RESULTS FROM PROGRESSIVE DECREASE IN GLOMERULAR FILTERATION AND THE LOSS OF TUBULAR FUNCTIONS.

CAUSES :

1-CHRONIC DESTRUCTIVE RENAL DUSEASES.

2-CHRONIC RENAL VASICULAR DISEASES.

3-OSBSTRUCTION TO THE URINE OUTFLOW.

4-CONGENITAL LESIONS .

5-METABOLIC CONDITIONS.

1-CHRONIC DESTRUCTIVE RENAL DISEASES INCLUDES:

1-CHRONIC PYELONEPHRITIS
2-CHRONIC GLOMERULONEPHRITIS
3-SYSTEMIC LUPUS ERTHEMATOSIS
4-BILATERAL RENAL TUBERCULOSIS
5-RADIATION NEPHRITIS

2-CHRONIC RENAL VASCULAR DISEASES :

1-MALIGENANT HYPER TENSION.
2-RENAL VEIN THROMBOSIS

3-OBSTERUCTION TO THE URINE OUTFLOW

1-THIS IS MANIFESTED BY BILATERAL HYDERONEPHROSIS AND OCCURS SECONDERILY TO URETHERAL OBSTERUCTION FOLLOWING :

1-ASTRICTURE

2-PROSTATIC ENLARGEMENT.

3-CARCINOMA OF THE CERVIX ,WHICH MAY ALSO OCCLUDE BOTH URETERS

4-CONGENITAL LESIONS AS :

1-POLY CYSTIC DISEASE
2-CERTAIN TUBULAR DEFECT MAY EVENTUALLY LEAD TO SEVER RENAL FAILURE

: - 5-METABOLIC CONDITIONS

1-HYPER CALCEMIA ,HYPOKALEMIA , AMYLOIDOSIS , GOUT
2-DIABETES MELLITUS (DIABETIC NEPHROPATHY)
ALL MAY LEADS TO SERIOUS RENAL DYSFUNCTION ESPECIALLY IF THEY PERSIST FOR LONG DURATION.

LABORATORY FINDINGS : INCLUDES :

1-DISTURBANCE IN WATER METABOLISM.

2-HYPONATREMIA.

3-MILD RETENTION OF THE POTASSIUM.

4-DISTURBANCES IN ACID BASE BALANCE .

5-DISTURBANCES IN CALCIUM AND PHOSPHORUS METABOLISM.

6-IMPAIRED GLUCOSE TOLERANCE AND INCREASED INSULIN RESISTANCE IS COMMON.

7-RETENTION OF WASTE PRODUCTS : UREA ,URIC ACIDS , CREATININE.

8-HYPERTENSION .

9-HEMATOLOGICAL EFFECTS

LABORATORY FINDINGS :

1-DISTURBANCE IN WATER METABOLISM : CLEARLY SHOWS :

***THE INABILITY OF THE FAILING KIDNEY TO CONCENTERATE THE URINE ADEQUATELY LEADS TO MODERATE POLYURIA ,WHICH IS CONSIDERED EARLY SIGNS IF RENAL FAILURE THE POLYURIA ALSO OCCURS DUE TO OSMOTIC OSMOTIC DIURESIS**

***POLYURIA IS ESPECIALLY MARKED IN CHRONIC PYELONEPHRITIS AS THE CONCENTRATING PART OF MEDULLA IS AFFECTED EARLY.**

***AMILD DEGREE OF DEHYDERATION OCCURS AND IS MANIFESTED BY THIRST.**

4-DISTURBANCES I ACIDBASE BALANCE :

PROGRESSIVE RENAL FAILURE IS ACCOMPANIED BY AMETABOLIC ACIDOSIS DUE TO RELATIVE FAILURE OF ACIDIFICATION OF URINE DUE TO IMPAIRENT IN AMMONIA PRODUCTION ,AND THE ABILITY TO SECRETE HYDROGEN ION (H+) IS IMPAIRED.

5-DISTURBANCES IN CALCIUM AND PHOSPHOROUS :METABOLISM

IN CHRONIC RENAL DISEASE:THERE IS APERSISTANT RETENTION OF PHOSPHATE AND SERUM PHOSPHATE RAISED

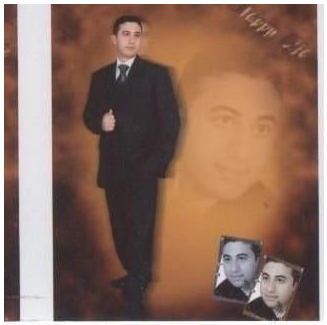
THE SERUM CALCIUM IS SOMEWHAT REDUCED AS AN INVERSE RESPONSE TO THE RAISED SERUM PHOSPHATE AND ALSO DUE TO IMPAIRED INTESTINAL ABSORPTION OF CALCIUM (BECOUSE OF VITAMINE D RESISTANCE AND DEFICIENCY)

SECOUNDARY PARATHYROID HYPERPLASIA MAY OCCUR ,THE PARA THORMONE MOBILIZES CALICIUM AND PHOSPHATE FROM THE BONES THEREFORE ,OSTEOMALACIA (RENAL RICKETS)OCCURRED.

9-HEMATOLOGICAL EFFECTS AND PATHOLOGICAL CHANGES:

SEVERE NORMOCYTIC NORMOCHROMIC ANEMIA IS COMMON IN THE LATER STAGES OF RENAL FAILURE . ANEMIA IS ATTRIBUTED TO THE IMPAIRED PRODUCTION OF ERYTHROPOITIEN,WHICH IS NORMALLY FORMED IN THE KIDNEY.

PURPURA ,WHICH IS PROBABLY DUE TO AQUALITATIVE DEFICIENCY IN THE PLATELETS.



Some of Important bacterial diseases

Written and presented by

Doctor

Mohammad Mahmoud Mohammad Youssef wehaba

Assistant Researher

Master of bacteriology ,immunology,mycology

Animal health research institue

Some of Important bacterial diseases

Streptococcus	pneumonia , pharyngitis , cellulitis
Staphylococcus	abscess of skin and other organs
Bacillus anthraces	anthrax
Clostridium	tetanus , gas gangrene , botulism

Coryne bacterium	diphtheria
Listeria	meningitis
Actinomyces	actinomycosis

Nocardia	nocardiosis
Neisseria	gonorrhea , meningitis
Haemophilus	meningitis
Bortedella	whooping cough
Legionella	pneumonia
Brucella	brucellosis
Francisella	tularemia

Pasteurella	cellulitis
Yersinia	plaque
Escherichia coli	urinary tract infection , diarrhea
Enterobacter	urinary tract infection
Serratia	pneumonia
Klebsiella	urinary tract infection
Salmonella	enterocolitis , typhoid fever

Shigella	**enterocolitis**
Proteus	**urinary tract infection**
Campylobacter	**enterocolitis**
Helicobacter	**gastritis , peptic ulcer**
Vibrio	**cholera**
Pseudomonas	**urinary tract infection , pneumonia**
Bacteroids	**peritonitis**

Mycobacterium tuberculosis , leprosy
Rickettssia (rocky mountain spotted fever , typhus , Q fever)
Chlamydia urethritis , trachoma , psittacosis
Treponema syphilis
Borrelia lyme disease
Leptospira leptospirosis
Mycoplasma pneumonia

THANK YOU

التصنيفات للبكتيريا المهمة طبيا

جامعة بنها

كلية الطب البيطرى

قسم البكتيريا والمناعة والفطريات

كتبهاونسقها مساعدباحث محمدمحمودمحمديوسف وهيبه

ماجيستير البكتيريا والمناعة والفطريات

Classification of medically important bacteria

1- RIGID, THICK- WALLED CELLS	2- FLEXIBL, THIN WALLED CELLS	3- WALL LESS – CELLS
	Leptospira	**Mycoplasma**

1- RIGID, THICK- WALLED CELLS

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)	B- NON-FREE-LIVING BACTERIA (OBLIGATE INTERACELLAR BACTERIA (as Rickettsia and Chlamydia)

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)		
1- Gram positive bacteria (stained appear blue colour)	2- Gram negative bacteria (not stained just Avery little appear red or pink colour)	3-Acid fast bacteria e.g T.B Mycobacterium

10- THE LEVELS OF CYSTATIN C ARE INDEPENDENT OF WEIGHT AND HEIGHT ,MUSCLE MASS,AGE, (OVER AYEAR OF AGE) ,SEX,AND FACTORS WHICH HAVE BEEN DOMENSTERATED TO AFFECT CREATININE VALUES,IN ADDITION,ARISE IN CREATININE DOES NOT BECOME EVIDENT UNTIL (THE GFR) HAS FALLEN BY APPROXIMATELY 50%.

11- MEASUTEMENTS CAN BE MADE AND INTERPRETED FROM ASINGLE RANDOM SAMPLE.

12- CYSTATIN C MAY IS USED AS AN ALTERNATIVE TO CREATININE AND CREATININE CLEARANCE TO SCREEN FOR AND MONITOR KIDNEY DYSFUNCTION IN THOSE WITH KNOWN OR SUSPECTED KIDNEY DISEASES.

13- IT MAY BE ESPECIALLY USEFUL IN THOSE CASES WHERE CREATININE MEASUREMENT IS NOT APPROPRIATE: FOR INSTANCE IN THOSE WHO HAVE LIVER CIRRHOSIS, ARE VERY OBESE, ARE MALNOURISHED OR HAVE AREDUCED MUSCLE MASS.

14- MEASURING CYSTATIN C MAY ALSO IS USEFUL IN THE EARLY DETECTION OF KIDNEY DISEASE WHEN OTHER PARAMETERS MIGHT STILL BE NORMAL, ESPECIALLY IN THE ELDERLY.

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<table>
<tr><td colspan="3">1- Gram positive bacteria (stained appear blue colour)</td><td colspan="2">2- Gram negative bacteria (not stained just Avery little appear red or pink colour)</td></tr>
<tr><td>a- Cocci
Staph
Strept</td><td colspan="2">b- Bacilli include : Spore forming and Non Spore Forming</td><td rowspan="4">a- Cocci like Nisseria</td><td rowspan="4">b- Bacilli include : Aerobic (Psudomonas) and Facultative Anaerobic (Straight – Curved) and Anaerobic bacteria (Bacteroides)</td></tr>
<tr><td rowspan="3"></td><td>Spore forming</td><td>Non Spore forming</td></tr>
<tr><td>Aerobic bacteria like Bacillus Anthracis
Anaerobic bacteria like Clostridia</td><td>non Filamentous like
Coryn bacteria,
Listeria and Filamentous Actinomyces</td></tr>
<tr><td></td><td></td></tr>
</table>

Facultative anaerobic

Straight			Curved
Respiratory bacteria Haemophilus	Zoonotic bacteria- Brucella , Pasteruella , Yersenia	Enteric bacteria E.coli , Salmonella, Shigella ,Klebsiella, Proteus	Campylobacter , Vibriocholera ,Hilicobacter

بعض التحصينات الإجبارية للأمراض الوبائية والكشف عنها داخل القطر المصرى
(كتبها ونسقها
دمحمدمحمودمحمديوسف وهيبه
بعد إرسالها المركزى) -

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

المرض المشتبه به	نوع العينة المراد العزل منها	المادة الحافظة	طريقة النقل المناسبة ودرجة الحرارة المطلوبة	المدة المسموح بها حتى الوصول إلى معهد الدقى
١-الحمى القلاعية (FMD)	حويصلة كاملة-بثرات- سائل الحويصلة	Glycerin buffer	فى ثلاجة نقل العينات محتوية على أكياس ثلج خاصة (4C) بحيث تكون كل عينة فى كيس منفصل	٢٤ ساعة
٢-حمى الوادى المتصدع (RFV)	دم على مانع تجلط –أجنة مجهضة (كبد-طحال-غدة ليمفاوية)	Universal transport medium	كما سبق	٢٤ ساعة
٣-الجلد العقدى (LSD)	عقدة جلدية – عقدة ليمفاوية – طحال	Transport buffer	كما سبق	٢٤ ساعة
٤-طاعون المجترات الصغيرة (PPR)	دم على مانع تجلط أثناء إرتفاع درجة الحرارة –بثرات من الفم – مسحات من العين والأنف والفم – أحشاء (الرئة غدد ليمفاوية – الأمعاء الطحال)	EDTA Heparin	كما سبق	٤-٦ساعات

٥-التسمم الدموى Pasteurella multocida	مسحات أنفية – أحشاء داخلية – دم –سيرم		كماسبق	٤-٦ساعات
٦-حمى الثلاث أيام Bovine ephemeral fever virus Rhabdoviridae	دم على مانع تجلط – غدة ليمفاوية - طحال	Preservative media PBS	كما سبق	٢٤ ساعة

٧- الإلتهاب الشعبى البقرى (IBR)	مسحات أنفية-غدة ليمفاوية-جزء من الرئة – جزء من القصبة الهوائية	Preservative media PBS	كما سبق	٢٤ساعة
٨-الإسهال المعدى (BVD)	مسحات أنفية-مسحات شرجية-سيرم-غدة ليمفاوية – أحشاءداخلية	Preservative media PBS	كما سبق	٢٤ساعة
٩-أمراض العجول الرضيعة pneumoenteritis,parainfleunza ,corona viruses,may be mixed with salmonella , mycoplasma, E,coli	عينات براز-سيرم-أحشاءداخلية-مسحات أنفية ودم	Preservative media PBS	كما سبق	٢٤ساعة

١٠-التسمم المعوى (Enterotoxaemia) Cholesteridial infections especially in sheep and goat farms need co vac 8	الأمعاءالدقيقة (مربوطة ولا يقل طولها عن ١٥ سم) –فص كامل من الكبد به الإصابة – كلية كاملة		كما سبق	٤-٦ساعات
١١-التفحم العضلى والأوديما الخبيثة Black Leg (black quarter) Malignant Edema Closteridium Choviae	العضلة المصابة المتفحمة – سائل إرتشاحى من مكان الإصابة		كما سبق	٤-٦ساعات
١٢-المرض الأسود Black Closteridium (Disease) Choviae	فص كامل من الكبد به إصابة –الأمعاء الدقيقة		كما سبق	٤-٦ساعات

١٣-باسيلارى هيموجلوبين يوريا (بول أحمر) (Bacillary Hemoglobinuria) Clostridium novyi type D	فص كامل من الكبد به الإصابة		كما سبق	٤-٦ساعات
١٤-تعفن الحافر (Foot Rot) Fusobacterium necrophorum	مسحات من الحافر المصاب بعد تنظيفه جيدا من الأنسجة الميتة	المسحات على ناقل (Cary-Blair Medium)	كما سبق	٤-٦ساعات
١٥- التسمم الغذائى	-الأعلاف (لا تقل العينة عن ٥٠-١٠٠جرام) -السيلاج (لا تقل العينة عن ٥٠-١٠٠ جرام)- المياة (لا تقل عن ٢٠٠ مل)		كما سبق	لا تزيد عن ١٢ ساعة بطريقة الحفظ المذكورة

ملحوظة هامة : البروسيلاوالإسبريجلوسيس يسببان عاصفة من الإجهاضات ومن الأمراض التى يجب تبليغ جميع السلطات الأمنية الغذائية ويكتب ويرفع بها تقرير مبين وتم نسيانها من المختصين العاليين

يجب الإفادة عن الموقف التحصينى لتلك الأمراض عند إرسال أى عينات للفحص

وكيل المعهد للتشخيص المعملى وصحة الأغذية

تحريرا فى : / /

يعتمد

أ.د

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

جامعة بنها

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معهد بحوث الصحة الحيوانية

مركز البحوث الزراعية

وزارة الزراعة

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

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(التوبة - ١٠٥)

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS

FAMILY: ENTEROBACTERIACEAE

THE DEFINITION OF ENTEROBACTERIACEA:

Intestinal bacteria describe large biochemically and antigenically related groups of organisms

FAMILY: ENTEROBACTERIACEAE

1-Some of them are pathogenic potential to man, animals, insects and plants.

2- It contains about 4 tribes, 12 genera on the basis of DNA relatedness.

3-Generally the members of family enterobacteriaceae is composed of gram negative rods which may be motile by peritrichous flagella or non-motile.

4- They are non -spore forming and not acid fast.

5- Most of them are non capsulated but some have capsules or microcapsules (slime layer).

6- Enterobacteriaceae members grow on the presence and the absence of free oxygen because facultative anaerobes have the presence of respiratory and fermentative metabolisms.

5- They grow well on ordinary media.

6- They are similar in the morphological and cultural characters.

7-Moreover all species contain the enterobacterial common antigen.

8- Biochemically they produce acid and gas due to the fermentation of glucose or different types of carbohydrates.

9- All species are catalase positive .

10-Reduce nitrate to nitrite.

11- Oxidase negative .

12- According to their ability to ferment lactose they were differentiated biochemically into two divisions :

1 - Lactose fermenting enterobacterial genera.

2-Non lactose fermenting enterobacterial genera .

This classification depends on the cultivation of the species of family enterobacteriaceae on one of the following differential selective media :

1-MacConkey agar (lactose-bile neutral red agar)

2-S.S media (salmonella- shigella agar)

3- Desoxycholate cirate agar

4- Hegkton media

Different genera of Enterobacteriaceae according to their ability to ferment lactose : Lactose fermenter and Non Lactose +(Reacted Fundamental Environment) fermenter

Lactose fermenter :

Genus	Habitate
E.coli	Intestine
Enteobacter	Intestine , water
Citrobacter	Intestine, water , soil
Yersenia	Intestine, water
Serratia	Intestine, water , ,soil

Non- lactose fermenter :

Genus	Habitate
Shigella	Intestine ,water, soil
Salmonella	Intestine , water , soil
Hafnia	Intestine, water,soil
Proteus	Intestine, water , soil
Morganella	As Proteus

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

WRITTEN AND PRESENTED BY DOCTOR
MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA .
MASTER DEGREE BENHA UNIVERSITY , ASSISTANT RESEARCHER,
ANIMAL HEALTH RESEARCH INSTITUTE
Agricultural Research Center

.THANK YOU .

GENUS: ESHCERICHIA
SPECIES: ESCERICHIA COLI (E.Coli)
Synonym: BACTERIUM COLI (E.Coli)

Definition: Escherichia coli is a normal inhabitant of the lower part of intestine of all warm blooded animals.

It is rarely present in stomach and in few numbers in the anterior part of intestine. Carnivores and omnivores usually harbor E.coli in greater number than herbivore.

Under certain conditions of stress and environmental factors may undergo to be potential pathogenic causing sever diseased conditions.

Specific diseases caused by enteropathogenic E.coli in different animal species:

Species	Disease	Symptoms
Childrens	Coli septicemia	**Diarrhea , death**
Newly born animals	Colibacillosis, white or calf scour	**Septicemia and rapid death**

Microscopic characters :

E.coli is coccobacilli to long rods (.6 x1.3M), arranged singly or in short chains especially in mucoid colonies where it had a slime layer of polysaccharides . it is motile and non – sporulated .

Cultural characters :

(Nutrient broth-Nutrient agar-Blood agar-MacConkey agar)

-In nutrient broth growth is rapid , gives regular turbidity within 12-18 hours at 37 C with heavy powdery sediment after 24 hours .

-On nutrient agar growth

1-smooth surface colonies occurs after 24 hours,

-The colonies are grayish in color , circular , convex 2-3mm in diameter .

 2- mucoid colonies appear due to the presence of slime layer and larger in size than smooth one .

-On blood agar the surface colonies are larger some are hemolytic and others are non-hemolytic

-On macConkey agar it gives round pink colonies, medium sized opaque colonies i.e lactose fermenter

(Viability And Resistance) :

-It may survive for weeks or months in contaminated water, dwellings in the animal houses in the presence of mucous or feces . Some strains resist dryness.

-It is susceptible to physical and chemical agents. E.coli is killed by heating at (55-60 C) for 30 minutes, within 10 minutes by autoclaving.

The Antigenic Structures Of E.coli include three antigens : 1-somatic antigens 2- capsular antigens 3- flagellar antigens . Detected by serological diagnosis

1-Somatic antigens (146 O- antigens)
2-Capsular antigens (91 K –antigens)
3- Flagellar antigens (49 H- antigens) :

1-Somatic Antigens : (146 O- antigens) :

E. coli strains have 146 O-antigens in their body . Each strain carries only one type of O-antigen . They are composed chemically of polysaccharide–phospholipids –protein complex. The O –antigens are designated by numbers as O1, O2, O3,O4 ,O5 – O 145 etc .

The O – antigens are heat stables.

2- Capsular antigens (91 K –antigens) :

These 91 k-antigens occur as a slime layer or microcapsule on most enteropathogenic E.coli strains.

Chemically they are composed of poly saccharides . k-antigens prevent agglutination between O-antigens and their specific homologous antisera.

The K – antigens are differentiated into

3 types according to their thermo stability as follows :

L-antigen : heat labile

A- antigen : heat stable

B- antigen : lipopoly saccharide destroyed by autoclaving

The K-antigen is very important in serotyping of E.coli as well as in the mechanism of pathogenesis as K 88 and K 99 .

These K 88 and K 99 mediate adhesion of the organism to the microvilli of the intestinal epithelial cells and enhance colonization .

3-Flagellar antigen (49 H-antigens) : There are 49 H-antigens which is composed of protein . frequently it is poorly developed .

It has weak role in serotyping of E.coli.

-Serotyping Identification :

For serotyping of E. coli , identification of O and K antigens is applied by slide agglutination test to determine its antigenic structures as O20/B6 , O55/B5 , O88/B4 , O111/B4 .

Toxins of E.coli (E.coli products) :

1-Enterotoxin 2- colicins

1-Enterotoxin:

This substances is produced by entero pathogenic strains of E. coli , and causes diarrhea in man and animals . Injection of E .coli enterotoxins into the ligated intestinal loops of rabbits induced swelling , oedema and accumulation of exudate in the injected loop. The enterotoxins are not produced by all E.coli strains but only the enteropathogenic.

E.coli enterotoxin is composed of two fractions:

a- Heat stable fraction.
b-Heat labile fraction.

a-Heat stable fraction : it is exteracellular in the culture . it is resistant to heating for 60 C but destroyed by autoclaving .
b- Heat labile fraction: it is inactivating by heating at 60 C for 30 minutes it is antigenic and occurred within bacterial cells . it could be neutralized by specific antisera .

2- Colicins :

It is E.coli products and composed chemically of protien . it has bactericidal activitiy . colicins productions is very important in differentiation of E.coli strains into :

Colicins positive E.coli.

Colicins negative E.coli.

Laboratory diagnosis:

- The isolation of E.coli may be of no value as E.coli are normal inhabitant of the intestinal tract, unless other seven factors are considered including:

1-Detailed history of the case, symptoms and lesions with differential bacteriological diagnosis on the other causative agents of digestive disorders .

2-Purity of the growth of E.coli on the primary cultures note the growth of lactose fermenting colonies on MacConkey agar .

3-Site of isolation from various organs especially from the parenchymatous organs as liver , spleen , mesenteric lymph nodes and lung tissues indicating colibacillosis.

4- Biochemically identification of isolated suspected E.coli the suspected E.coli colonies as lactose fermenter on macConkey media can be tested biochemically And must be differentiated from the other lactose – fermenter species by the following reactions (Indol , Methyle Red , VP , Citrate , Urea Hydrolysis , H2S, Motility , KCN) (Voges proskauer)(potassium cyanide)

E.coli are motile and Indol and Methyl Red Positives Only .

5-Serotyping of E. coli if the isolating colonies, are biochemically typical of E.coli, Serotyping must be performed using polyvalent O, K anti-sera to determine the antigenic structure of the isolated strain of E.coli.

6-Final diagnosis depends mainly upon the isolation of E.coli from various fresh organs in case of septicemia of the same antigenic structure.

7-It is very essential that the same serotype of E.coli must be isolated from feces or intestinal content as well as the internal organs of the same case .

WRITTEN AND PRESENTED BY
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THANK YOU

Benha University
Faculty of Veterinary Medicine
Bacteriology, Immunology, Mycology Department

Occurrence of food borne pathogen in some types of soft cheese in local market

A Thesis Submitted to
Faculty of Veterinary Medicine
Benha University

By

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(BVSc., Kafer Elsheikh University, 2003) For the degree of M.Sc.
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بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

(قالوا لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم)

Acknowledgement

First of all, I wish to express my deep thanks to Allah Almighty who has endowed his help and blessings on me to have the present work achieved in such condition.

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Thanks, you all for your patience and support

VITA

The Author Was Born In September 9, 1981 In El – Behera Governorate, Egypt.

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His Undergraduate And Professional Educations was Completed In Faculty Of Veterinary Medicine, Kefir El – Sheikh University From Which He Received a BVSc. Degree In 2003. Very Good. The Seventh.

He works at animal health research institute, Tanta branch, Connected To The Main Institute In The Dokki -Giza -Cairo Under Super Visions Of Agricultural Researches Center Under Laws Of Universities Learning And Researches Qualifications.

Dedications

My Father
My Mother
My Sister
My Brother
My Uncles
My Family

Always all support religious forever.

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1. INTERODUCTION

Food safety is considered one of the most common urgent matters in the food industry worldwide. Spoilage of the food products with foodborne pathogens receive a special concern among food producers, investigators and customers. Consequently, producing a safe food represents one of the most imperative urgencies in the food processing (Friedman *et al.*, 2002; Mohamed *et al.*, 2013).

The outbreaks of foodborne illnesses caused by *E. coli* have been studied previously in developing countries after ingestion of milk products such as old-style cheese which is considered the major source of various types of pathogenic bacteria (Elhaida and Mohammed, 2013).

E. coli **is one of the major significant bacteria, which has a bad effect on both human and animal species. Thus, this type of bacteria can deteriorate the milk particularly raw milk and other milk products as a result of poor hygienic measures (Lara *et al.*, 2016; Garbage *et al.*, 2016).**

Prevention of foodborne illness is a global concern that affects all aspects of food production, including dairy. Dairy cattle are known reservoirs for zoonotic bacteria and milk and milk products have been implicated in outbreaks of foodborne illness (Wells *et al.,* 1991; Zhao *et al.,* 1995; Wallace, 1999). These outbreaks are generally caused by improperly pasteurized products, post pasteurization contamination, or by raw milk and raw milk products.

pathogenic *Escherichia coli* are also present in dairy production environments and have been associated with milk and milk products (Ryser, 2001; Watseka and Kinema, 2003). Although milk can be contaminated with bacterial pathogens via shedding in the udder, these zoonotic pathogens are not commonly associated with mastitis. Fecal contamination is considered to be the most common route of bulk tank milk contamination with these organisms.

Escherichia coli **live commensally in the gastrointestinal tract of most mammals, including humans, without causing disease. However, a small fraction of *E. coli* are human pathogens and have been implicated in foodborne illness with increasing frequency over the last 2 decades. Ingestion of enteropathogenic *E. coli* (EPEC) can result in a mild gastrointestinal disease that is relatively self-limiting but a subset of the EPEC, can a serious potentially fatal illness (O'Brien and Kipper, 1998). The most common human clinical EPEC isolated in the United States is O157:H7 (Canavalia *et al.,* 2001; Watseka and Kinema, 2003).**

E. coli **is classified into six pathotypes: enteroaggregative, enterohemorrhagic Shiga toxin-producing *E. coli* (STEC), enteron invasive, enteropathogenic, enterotoxigenic, and diffuse adherent (Jafari *et al.*, 2012). lethal STEC which cause severe illnesses were also detected (Boutin *et al.*, 2007).**

Previous studies indicated that STEC represents one of the most significant pathotypes which lead to foodborne illnesses compared with other types *E. coli* (Brett *et al.*, 2003; Kaufmann *et al.*, 2006). In human, the pathogenic effect of STEC nearly due to its ability to produce certain types of cytotoxins for example Shiga toxins (*stx1*), and intimin (*eae A*) virulent genes (Slaney *et al.*, 2009 and Assupol *et al.*, 2015).

Cheesemaking is a major industry worldwide, and much of it is still practiced on a relatively small scale with only basic hygiene practices (Kostas *et al.*, 2010; Dias *et al.*, 2012).

Among the dairy products, cheese is considered one of the most public sources of vital nutrients (*e.g.* vitamins, minerals and proteins) which represent the main part of healthy food (López-Expositor *et al.*, 2012). In Egypt. Moreover, Kariesh cheese is a prevalent type of cheese which contains a higher protein content with a small amount of fats (Hamad, 2015).

Nevertheless, cheese is considered a safe food for human being, sometimes its deterioration by various types of foodborne pathogens may take place. Enteropathogenic *E. coli* (EPEC) are considered the most common bacteria isolated from cheese. EPEC also cause high morbidity and mortality rates among young and old people (Kostas *et al.*, 2010).

Kariesh cheese are the most popular soft cheese in Egyptian cities and Arabian countries, similar to Domiati (Brown, 2004; Abd-Hamid, 2012; Hegazi *et al.,* 2012). It is an acid coagulated fresh cheese, made from skimmed buffalo's or cow's milk or a mixture of both with soft composition, white curd and slightly salty (Francois *et al.,* 2004; Abou-Donia , 2008), but mostly produced from skimmed buffaloes' milk and its manufacturing depends on acid coagulation by the action of rennet & lactic acid bacteria

Escherichia coli **is commonly present in the intestinal tracts of humans and animals, and its detection in processed foods is an indication of fecal matter contamination. It also indicates the inadequate controls of the environment of the cheeses processing units, the personals hygiene's of the food handlers, and the poor qualities of the finished products (Kostas *et al.*, 2010; Dias *et al.*, 2012).**

Because the occurrence of an Outbreaks of a foodborne disease is caused by Enteropathogenic *E. coli* and is associated with the consumptions of soft ripened cheese (Maurier *et al.*, 1973), the presence of these microorganisms in cheese has taken on a major significance.

Shiga toxins–producing *E. coli* (STEC), also known as serotoxins-producing *E. coli* (VTEC) or Enterohaemorrhagic *E. coli* (EHEC), have been known as a group of highly pathogenic *E. coli* strains producing one or more Shiga toxins (Monaghan *et al.*, 2011).

STEC represent a hazardous public health problem worldwide causing various human gastrointestinal tract diseases, including watery or bloody diarrhea and might develop a life-threatening disease, such as hemorrhagic colitis (HC), Thrombotic Thrombocytopenic Purpura (TTP) and Hemolytic Uremic Syndrome (HUS) (Pennington, 2010).

STEC strains produce two powerful phages encoded cytotoxins causing tissue damage in humans and animals, called Shiga toxins or serotoxins (*Stx1/VT1* and *Stx2/VT2*), which are the common features and main virulence factors of STEC and are directly correlated with human pathogenicity. (Lindgren *et al.*, 1993).

Stx1 **is one of the most powerful toxins, and the toxin producing strains are usually associated with more severe infections (Monies *et al.*, 2004 and Giles, 2007).**

STEC serotypes including O157:H7 STEC and non-O157. The O157:H7 STEC strain is considered one of the most important of all known food-borne pathogens because of the severity of associated illnesses produced by it and also, the apparent low infective dose (of less than 10 cells) (Bach *et al.*, 2002, Blanco *et al.*, 2003).

The most common, non-O157 strains associated with disease in humans including O26, O91, O103, O111, O128, O113 and O145 are responsible for up to 20 to 50% of all STEC infections (Johnson *et al.*, 2006).

There is a lack of information concerning the stress responses of many non

O157:H7 STEC causing human illness under stress conditions as most of these studies only focused on O157:H7 (Allen *et al*. 2008; Dong and Schell horn 2009; Vanaja *et al*. 2010). *eae* Agene it an intimin secretion gene that enhance attachment and colonization of *Escherichia coli* in the intestinal epithelium leading to sever cellular destruction and severe infections and sever symptoms like profuse diarrhea and ulcerative colitis (*Ibrahim et al.*, 2019*).*

Damiati cheese is one of soft pickled cheese and makes up about 75% of the cheese produced and consumed due to its nutritional value, convenience and have rich taste, strong sharp flavor as well as smooth body and texture.

Tallaga cheese is the most popular local type of packaged or unpackaged soft cheeses by all socioeconomic classes in Egypt due to its nutritional values, conveniences and good tastes, clean pleasant creamy low salty taste with a spreadable mellow soft body. It is a product closely related to Domiati cheese and mainly ready for consumption within one month of storage at refrigerator temperature.

Cheeses are more susceptible to spoilage due to microbial contamination. The source of contamination include both intrinsic factors (nutrients, water activity, PH, temperature, inhibiting factors produced by starter cultures and nonstarter microorganisms, (competitive microflora, etc.) and extrinsic factors (microbial quality of raw milk, production phases, ripening and packaging conditions, presence of chemical preservatives), cheese vat, cheese cloth and curd cutting knife, production room air and storage room(Hosni *et al.,*2011).

The improperly cleaned plants, equipment and food handlers are the main sources of contaminations to the cheese. Consumption of contaminated cheese with pathogenic microorganisms and their toxins can be used serious food borne problems to threat public health and big economic losses. But the most traditional cheeses are usually produced under poor hygienic conditions with different manufacturing technologies in local dairy plants.

***Escherichia coli* (*E. coli*) is one of normal inhabitant microorganisms of large intestine in human and warm-blooded animals. The main source of *E. coli* in raw milk and milk products is fecal contamination together with poor hygienic practices (Garbage *et al.*, 2016; Lara *et al.*, 2016). However, certain serotypes of *E. coli* have acquired some virulence-associated genes that enable them to cause intestinal or Extra-intestinal diseases (Hodson *et al.*, 2017).**

E. coli **is generally used as an indicator of direct or indirect fecal contamination and the possible presence of enteric pathogens in milk and dairy products (Kurnai and Johnson, 2001).**

These serotypes that cause enteric infections are generally called diarrheagenic *E. coli* strains, and their pathogenesis is associated with a number of virulence attributes, which vary according to pathotype.

Currently, diarrheagenic *E. coli* strains are classified into seven main pathotypes based on their distinct virulence determinants and pathogenic features, including enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohemorrhagic *E. coli* (EHEC), shiga toxin-producing *E. coli* (STEC), enter invasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAEC) and diffusively adherent *E. coli* (DAEC) , Uropathogenic *E. coli*

Uropathogenic *E. coli* (UPEC) is the most frequent pathogen responsible for up to 80% of UTIs have special virulence factors, including pili or fimbriae, especially type 1 which have the structural rule gene called *Fim H* gene which mediate attachment to uroepithelial and vaginal cells, resistance to human serum bactericidal activity, hemolysins production.

virulence factors of UPEC strains have a significant role in development of UTIs. The most virulence factor dependent upon the UPEC include adhesions type 1 fimbriae encoding by *Fim H* gene (Rutter *et al.*, 2006; Xia *et al.*, 2010) (Zahren et al 2014).

The serotypes associated with illness in humans include members of O serogroups 26, 91, 103, 111, 113, 121, 145 and 157 (Karbala *et al.*, 2010; Pizarro *et al.*, 2014).

As soft cheeses (Kariesh, Talega and Damietta) collected from different localities from Gharbia governorate may subjected to contaminations with *E. coli* and may constitute public hazard.

E. coli **constitutes publics hazards of living creatures especially humans (likes meningoencephalitis, pyelonephritis, hepatitis, pneumonitis, pleurisy, pericarditis, peritonitis, osteomyelitis, appendicitis).**

Therefore, this work was planned to covers the followings points:

1-Isolations, biochemicals and serologically identifications of pathologically pathotypes of Escherichia coli from Kariesh, Tillage and Damietta types of cheeses in el _Gharbia governorate.

2-Molecular identifications of isolated bacteria (Bacterium Coli) using PCR Techniques)

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1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
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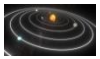

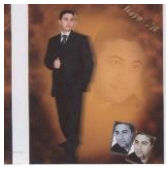

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3. Luxembourg
4. Switzerland
5. Slovenia
6. Croatia
7. Bosnia and Herzegovina
8. Czechia
9. Slovakia
10. Austria
11. Hungary
12. Serbia
13. Moldova
14. North Macedonia
15. Albania
16. Cyprus
17. Lebanon
18. Guinea-Bissau
19. Guinea
20. Ghana
21. Togo
22. Benin
23. Cameroon
24. Equatorial Guinea
25. Rwanda
26. Cambodia
27. Panama
28. Malawi
29. Liechtenstein
30. Montenegro
31. Kosovo
32. Palestinian Territories
33. St. Vincent & the Grenadines
0 2,000 4,000 Miles
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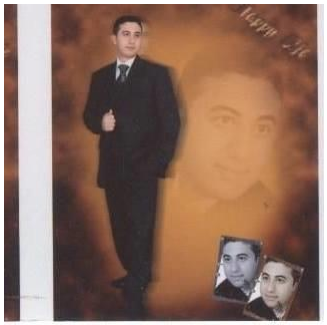

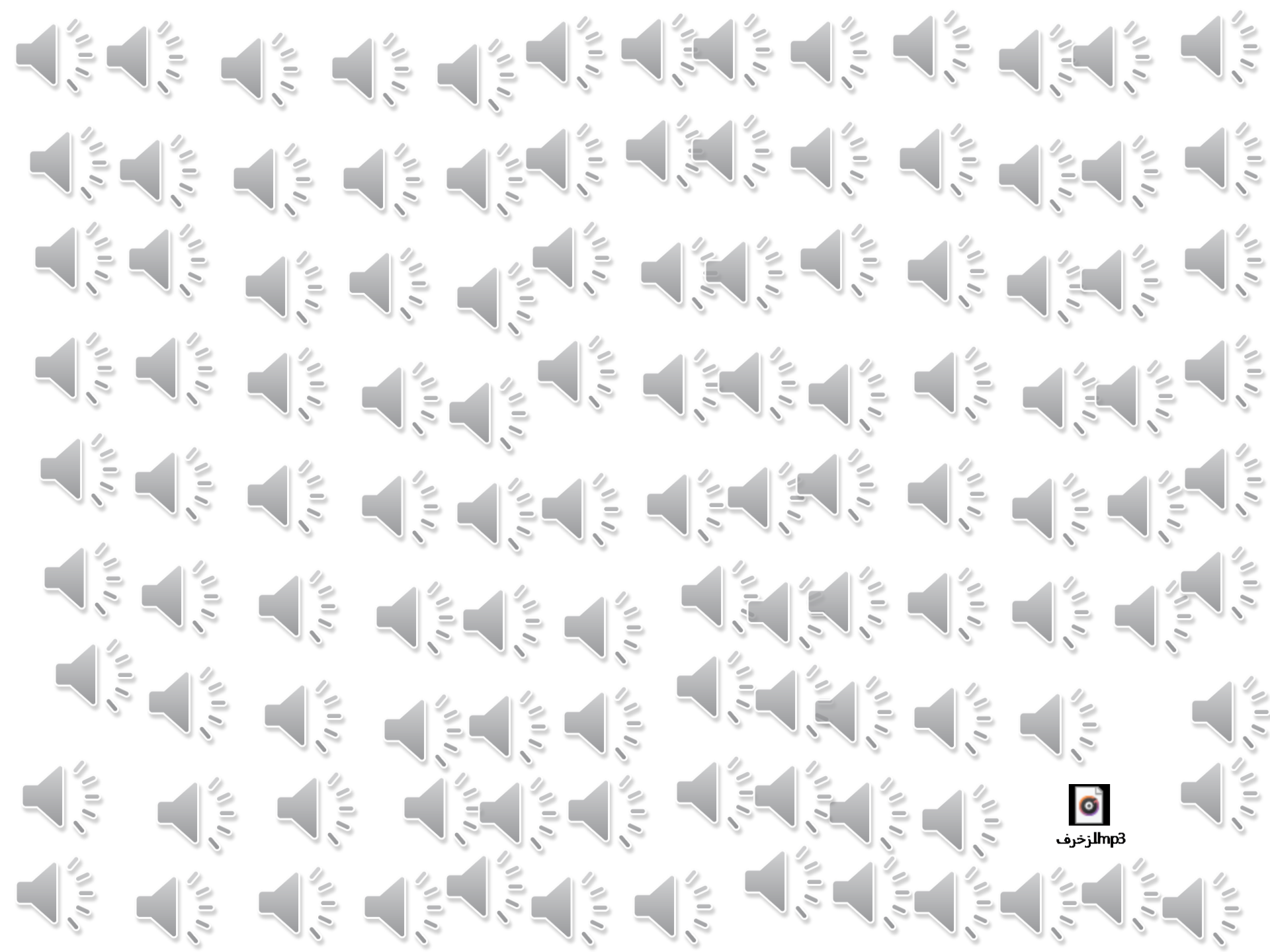
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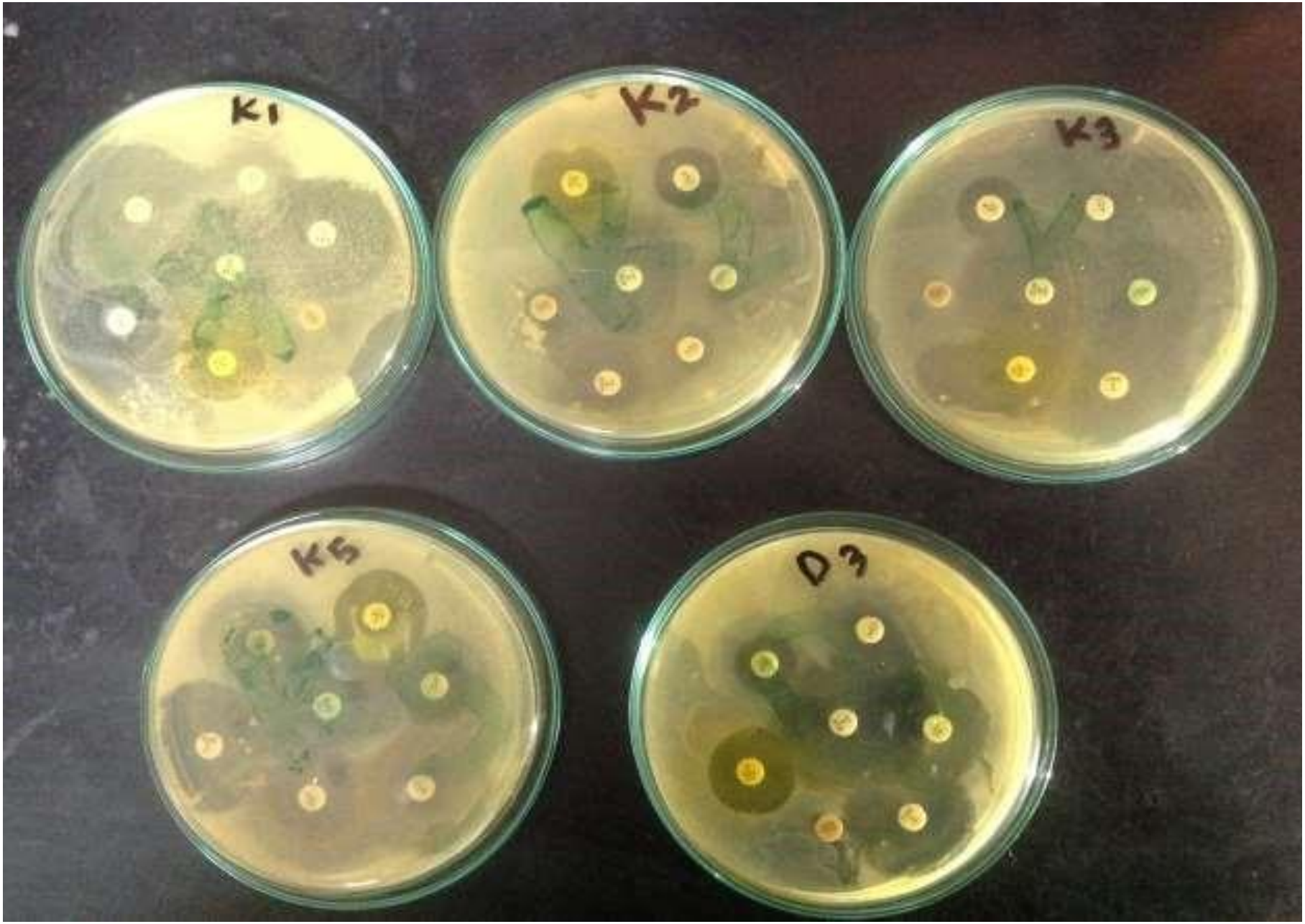
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K2
K3
K5
D3

What is the difference between PhD and Doctor of Philosophy؟

The PhD, also known as the Doctor of Philosophy ، is a research degree, which is one of the most common types of doctoral degrees ، and is awarded to graduates in many different fields

What is a PhD in philosophy called

A Doctor of Philosophy (PhD, Ph.D., or DPhil; Latin: philosophiae doctor or doctor philosophiae) is the most common degree at the highest academic level awarded following a course of study.

How long is a PhD in philosophy?

five year

The Doctor of Philosophy (PhD) is a five year program that will prepare you for an academic career in research and teaching in philosophy.

Is a PhD in philosophy difficult? Earning a PhD in philosophy takes hard work and perseverance. The process of getting your PhD in philosophy varies depending on the university and program. However, almost all programs will require coursework, intense research, and a completed dissertation. الرسالة

Is a doctorate better than a PhD?
Is a PhD Higher Than a Professional Doctorate? No, a PhD is not a higher degree than a professional doctorate. If you're wondering what's better than a PhD, both PhDs and professional doctorates are the highest level of college education that can be earned. Both degrees are doctoral degrees.

How do you address a Doctor of Philosophy?

D. When you are addressing a person with a doctoral degree, it is considered more polite to use the title Dr. or the academic abbreviation PhD with the person's name, instead of the simple courtesy titles Mr. or Ms.

What job does a Doctor of Philosophy do?

Professionals with a doctorate in philosophy often begin careers as professors at colleges and universities. Philosophy doctoral degrees may also lead to administrative roles in higher education or leadership positions in business or government settings.

What do doctors of Philosophy do?

What is a Ph. D.? A Ph. D., or Doctor of Philosophy, is a high-level degree earned after a period of three or more years of graduate-level study, culminating in the creation, submission, presentation and defense of a research dissertation.

بلغت ذروتها –إستسلام -الرسالة

Do Philosophy majors make any money?

While the starting salary is near the middle of all majors, the average mid-career salary of philosophy majors quickly rises to $81,200.00, outperforming Finance, International Relations, Marketing, Business Management, Communications, Nursing, Biology, Health Care Administration, and many others.

التخصصات-

Why is it called Doctor of Philosophy? Studies in what once was called philosophy are now classified as sciences and humanities. Hence, most disciplines, other than few like medicine and law, have their roots in Philosophy. Accordingly the name of degree awarded is Doctor of Philosophy.Jun 30, 2020

التخصصات

Is there a demand for philosophy professors?

Philosophy professor salary and job outlook The BLS predicts employment opportunities for philosophy and religious studies postsecondary teachers may increase by 7% from 2019 to 2029.Aug 25, 2021
BLS BASIC LIFE SUPPORT

How To Become a Philosophy Professor in 6 Steps (With Tips)

If you're curious about answering big life questions and discussing different viewpoints with others, becoming a philosophy professor may be a great career for you. Philosophy professors spend their time teaching, researching and completing administrative work at a college or university. Understanding the process to become this type of professor can help you determine if this is a career you are interested in pursuing. In this article, we discuss what a philosophy professor is, share some of their job duties and provide steps for how to become a philosophy professor.

A Ph. D., or Doctor of Philosophy, is a high-level degree earned after a period of three or more years of graduate-level study, culminating in the creation, submission, presentation and defense of a research dissertation

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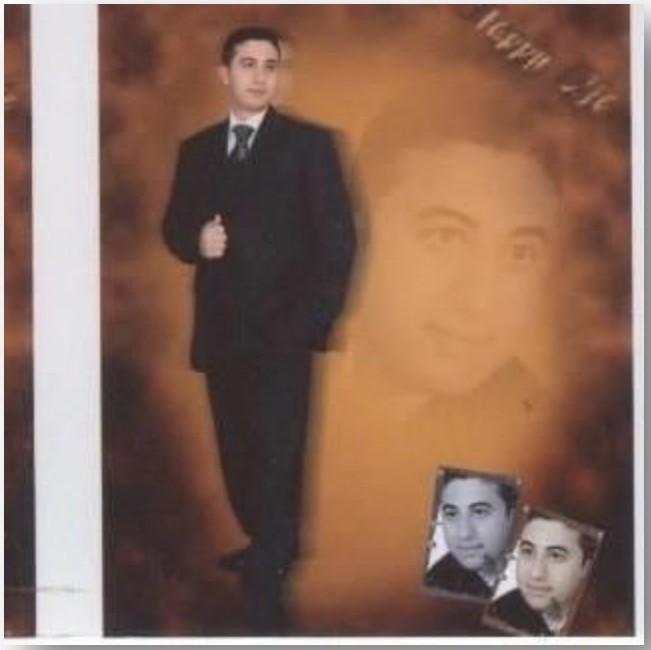


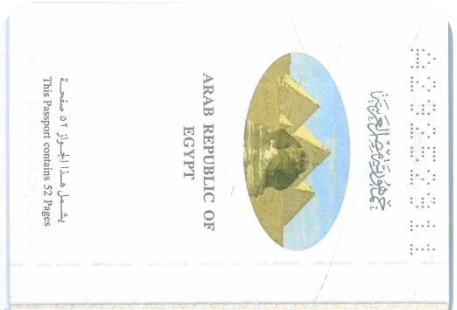
Bacteria

Bacteria, also called germs, are microscopic organisms not visible with the naked eye. Bacteria are everywhere, both inside and outside of your body.

Bacteria are small single-celled organisms. Bacteria are found almost everywhere on Earth and are vital to the planet's ecosystems. Some species can live under extreme conditions of temperature and pressure. The human body is full of bacteria, and in fact is estimated to contain more bacterial cells than human cells.

Bacteria are mostly free-living organisms often consisting of one biological cell. They constitute a large domain of prokaryotic microorganisms. Typically a few micrometres in length, bacteria were among the first life forms to appear on Earth, and are present in most of its habitats.

Most bacteria in the body are harmless, and some are even helpful. A relatively small number of species cause disease.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MOHAMMAD MAHMOUD MOHAMMAD YOUSSEF WAHIBA
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VETERINARIAN
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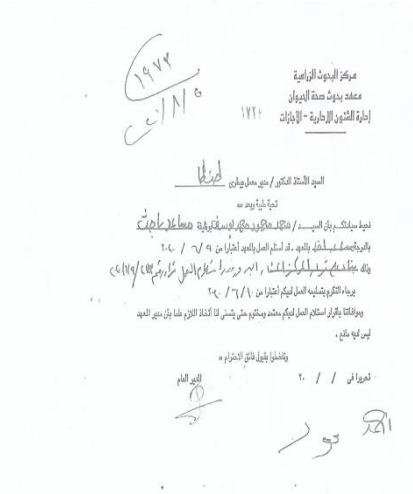
جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتاريخ : ١٩٨١/٩/٩
الجنسية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٠٢١/٤/٢٧
تاريخ اعتماد مجلس الجامعة : ٢٠٢١/٥/١٩
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بقرار رقم ١٦٩ بتاريخ ٢٠٢٠/١٠/٥
المختص رئيس القسم وكيل الكلية للدراسات العليا عميد الكلية
مستخرج من بيانات مركز الشهادات الموحدة بتاريخ ٢٠٢١/٦/٢٠

جامعة
كفر الشيخ
شهادة تخرج
063342
تشهد كلية الطب البيطري
أن / محمد محمود محمد يوسف
المولود فى البحيرة بتاريخ ١٩٨١م
قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية
فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً
مجموع تراكمى ٢٤٧٥ / ٣٣٠٠ النسبة المئوية ٧٥,٠٠%
وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م
ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا
الموظف المسئول المراجع مدير شئون الطلاب عميد الكلية
تحريراً فى ٢٠١٢/٥/٢٢م


مركز البحوث الزراعية
معمل بحوث صحة الحيوان
إجازة الشئون الإدارية - الإجازات
السيد الأستاذ الدكتور / مدير معمل بيطرى
نحية طيبة وبعد ،،
يرجاء التكرم بتشابه العمل لديكم اختيارا من ٢٠ / ٦ / ٢٠
وبرفائنا بالقرار استلام العمل لديكم منتدبا وبستحوم حتى يتسنى لنا أخذذ القرار فضا بان مدير العديد
ليس لديه مانع .
وتفضلوا بقبول فائق الاحترام ،،
تحريراً فى / / ٢٠
المدير العام

جمهورية مصر العربية
بطاقة تحقيق الشخصية
محمد
محمود محمد يوسف وهيبه
ش العوض -كفر الزيات
مركز كفر الزيات - الغربية
٢٨١٠٩٠٩١٨٠٠٥١٦
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طبيب بيطرى
معهد بحوث صحة الحيوان بطنطا
ذكر مسلم أعزب
البطاقة سارية حتى ٢٠٢٥/٠٧/٢٣
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وحده مرور دمنهور
رخصه قياده خاصه
١١٣٦٢٢
جمهورية مصر العربية
وزارة الداخلية
مرور البحيره
محمد محمود
محمد يوسف
مصرى
بكالوريوس طب بيطرى
ش طور سيناء ش الشيخة
دمنهور.

Morphology

Bacteria are complex and highly variable microbes. They come in four basic shapes: spherical (cocci), rod-shaped (bacilli), arc-shaped (vibrio), and spiral (spirochete)

Cell wall

Eubacteria are enclosed by a cell wall. The wall is made of cross-linked chains of peptidoglycan, a polymer that combines both amino acid and sugar chains. ... Some types of bacteria have an additional layer outside the cell wall. This layer is made from lipopolysaccharide (LPS), a combination of lipids and sugars.

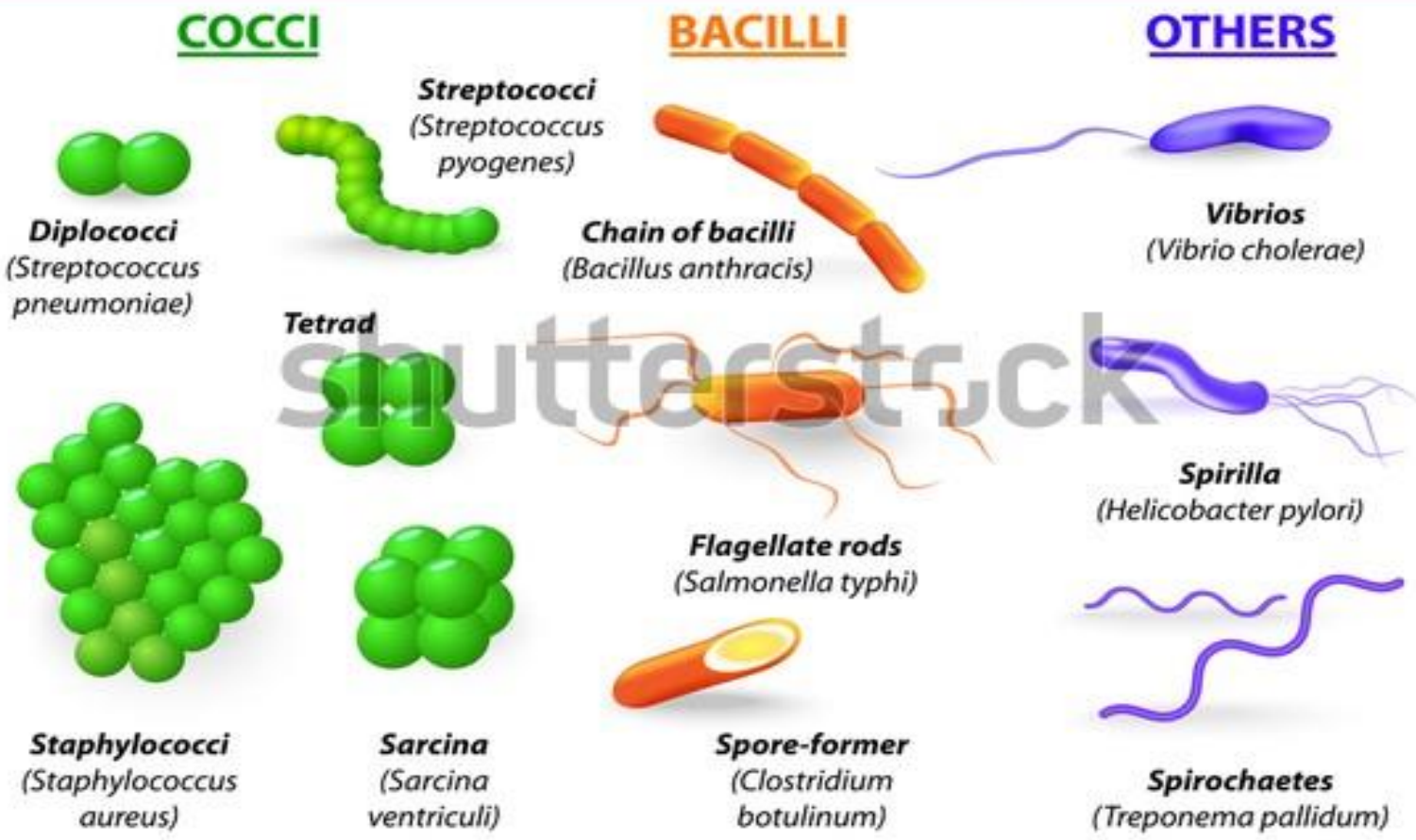
Nutrition

Nutrition. A great many of the most familiar eubacteria are heterotrophs, meaning they must take food in from outside sources. Of the heterotrophs, the majority are saprophytes, which consume dead material, or parasites, which live on or within another organism at the host's expense.

Treatment

Antibiotics are the usual treatment. When you take antibiotics, follow the directions carefully. Each time you take antibiotics, you increase the chances that bacteria in your body will learn to resist them causing antibiotic resistance. Later, you could get or spread an infection that those antibiotics cannot cure.

SHAPES OF BACTERIA



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SHAPES OF BACTERIA

SPHERICAL (COCCI)

Streptococcus pneumoniae

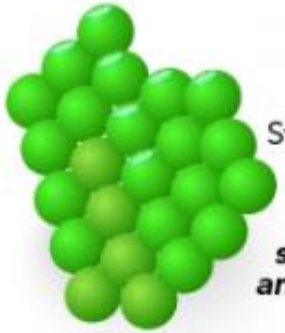
causes Pneumonia

Streptococcus pyogenes

causes Strep throat

Micrococcus luteus

causes armpits to stink



Staphylococcus aureus

can cause sinus infections and food poisoing

ROD-SHAPED (BACILLI)

(Bacillus anthracis)

causes Anthrax

(Salmonella enterica)

causes Typhoid

(Clostridium botulinum)

causes Botulism

SPIRAL-SHAPED (AND OTHERS)

(Vibrio cholerae)

causes Cholera

(Helicobacter pylori)

can cause stomach ulcers

(Treponema pallidum)

causes Syphilis

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BACTERIAE

Lectures Written
And Presented By
Doctor
Mohammed Mahmoud Mohammed Yousef Wehaba
Stipulated Course Of Bacterial Genatics Arranged By Department Of Veterinary Bacteria,immunology ,Mycology to pass exams
department ,benha university

SOME GENATIC WORDS

1-dalton الذرة

2-nicked مكشوف

3-integrated اندماجى و موحد ومستكمل

4-off spring سلالة ونسل

5-inter breeding تهجين ومزج السلالات

6-alternatively إرادى و طوعى

7-um جزء من المليون من المتر

8-auto باد ئة

9- vector ناقل

10-supplementary اضافى - ملحق

11-temperate معتدل - غير متطرف

12-lytic انحلالى - انهيار

13-autonomously مستقل – إستقلالى

14-dissemination إنتشار

15-transduction

16-lysogenic مستذيب - مسبب للتحلل

17-prophage طور أولى - لاقم أولى

18-conversion تحويل

19-confer يحدث – محادثة - يحول

20-phenotypic النمط الظاهرى

21-progeny أنسال

22-competence إستحقاق –جدارة - أهلية –إجادة

23-homology علم الشباة – تشابه تركيبى - تماثل

24-defective معيبة - بها عيوب

25-complement متمم - مكمل

26-inversion إنعكاس - إستقلاب

27-duplication مضاعفة

28-extensive واسع النطاق

29-substitution إستبدال

30-nucleases هو إنزيم يعمل على تحلل الحمض النووى الريبوزى منقوص الأكسجين بواسطة فك الروابط الموجودة داخل النيوكليوتيدات المتجاورة

31-recombination إتحادات جديدة والتوليف الجينى وإعادة التركيب وعودة الإلتئام

BACTERIAL GENETICS

BACTERIAL GENATICS OF MEDICAL IMPORTANCE OF BACTERIAL CELL

1-VIRULENCE
2-PATHOGENICITY
3-ANTIBIOTIC RESISTANCE

DETERMINED BY GENETIC INFORMATION IN THE CELL GENOME

BACTERIAL GENETICS

: is the study of how genetic information is transferred, either from: a particular bacterium to its offspring or between interbreeding lines of bacteria, and how that genetic information is expressed

THE GENETIC INFORMATION IS NORMALLY ENCODED BY THE SPECIFIC SEQUENCE OF NITROGENOUS BASES

Adenine A Guanine G Thyamin T Cytosine

Gene • **A sequence of DNA carrying codon**

Codon • **The genetic information is stored**

WHAT IS DEFINITION OF A DNA MOLECULE :

1- A DNA IS COMPOSED OF TWO CHAINS OF NUCLEOTIDES WOUND TOGETHER IN THE FORM OF ADOUBLE HELIX

2- EACH CHAIN HAS ABACK BONE OF DEOXYRIBOSE AND PHOSPHATES RESIDUES ARRANGED ALTERNATIVELY

Sources of genetic information in bacteria :

1-Bacterial Chromosome

2-Plasmid

3-bacteriophages

4-Transposons

Bacterial Chromosome

- Bacterial nucleoid contains a circular chromosome of double stranded DNA.
- About 1000 um long when straightened.
- Prokaryotic cells are haploid " has single chromosome copy

Plasmid

- Circular extra-chromosomal DNA molecules, present in cytoplasm of bacteria.
- Capable of auto-replication.
- Can transfer genes from one cell to another.
- Act as Vectors in genetic engineering.
- Can also present in yeasts

Plasmid

❖ carry supplementary genetic information coding for beneficial properties as antibiotic resistance

- Not essential for normal growth and reproduction of Bacteria.
- but may confer survival advantages “ antibiotic resistance” , toxin production

Plasmid

- Plasmid can be integrated with chromosomal DNA.
- Episomes: integrated form of plasmid with DNA.

Plasmid can transfer from cell to cell by : 1- Transduction 2- Transformation 3- Conjugation

- **Widely used for cloning DNA**

Medical importance :

1- Drug resistance: R plasmid (resistance to (antibiotics) 2- Virulence: Enterotoxin production 3- Antimicrobial agents: as col plasmid Production of bacteriocin 4- Sex –factor: F plasmid (fertility factor)

A third source of genetic information in a bacterial cell can be provided by the presence of certain type of bacterial viruses:

BACTERIO PHAGES

1-ARE VIRUSES THAT PARASITIZE BACTERIA AND CONSIST OF NUCLIEC ACID CORE AND PROTIEN COAT

2-APHAGE PARTICLE MAY HAVE AT ITS CORE BESIDE ITS OWN NUCLIEC ACID AND ASEGMNT OF THE HOST DNA

Viral genome ss single stranded DNA or RNA may be linear or circular

- **They are unable to multiply in the absence of their bacterial host.**
- **Lethal to the host cell.**

Phage "life" cycles

Virulent/ Lytic cycle

➢ After a large number of progeny are built up inside the host, the bacterial host ruptures and phages are released.

Phage "life" cycles

Temperate/ Lysogenic cycle ➢ Occur integration of bacteriophage nucleic acid into host bacterium's genome.

➢ The Prophage " genetic material of bacteriophage" is transmitted to daughter cells at each subsequent division.

➢ Then a later events as (UV radiation or presence of chemicals) can release them, with proliferation of new phages via lytic cycle.

➢ Controlled replication without causing lysis called lysogeny .

➢ In this case bacteriophages becomes temporary part of the total genetic information .

Functions of Bacteriophages

1- Gene transfer: Transfer of genetic information from one bacterial strain to another by transduction.

2- Lysogenic conversion: In which phage s own DNA confers new properties to the cell. (Toxignicity of Coryn. Diphtheriae).

3- Bacterial typing : Phage typing is one of the methods used to identify bacteria

4- Genetic engineering.

Variations in Bacteriae

1-Phenotypic Variations

2-Genotypic Variations caused by two ways:

1-Gene transfer 2-Mutation

1-gene transfer via this processes :-

A- Transformation

b-Transduction

d-Conjugation

2-Mutation leads to two Variations :

1-Phenotypic Variations .

2-Non Phenotypic Variations.

Phenotypic variation Any change under the influence of environment ✓ Reversible ✓ Unstable

✓ Non heritable

Such as : loss of flagella

loss of cell wall

change of colony appearance

(smooth, rough)

Genotypic variation ✓ Irreversible ✓ Stable ✓ Heritable due to changes in nucleotide sequences

mutation

 The bacterial genome is normally identical in all the progeny Due to asexual binary fission (bacterial reproduction). Therefore DNA replication is very accurate
 But rarely inaccuracies produce a slight altered nucleotide sequence in one of the progeny cell Called mutation (Stable “Irreversible” and Heritable ” Passed to subsequent generations”)

Mutation can occur in 2 ways :

A-Spontaneously Rare 1 - 10^6 Change of DNA .

B-Induced by using certain chemical or physical agents (mutagens):

1- U.V rays 2- X- rays

3- H_2O_2 .

TYPES OF MUTATION

1-Multisite mutation 2- point mutation

1-Multisite mutation:

affecting one or very few Extensive chromosomal rearrangements

Inversion one to another

one or more Duplication

2- point mutation :

-Extensive - substitution - deletion

Types of mutation Mutation involving extensive chromosomal rearrangements such as :

Normal (wild type) A b c d e f g
1- inversion a b f d e c g
2- duplication a b c b c d
3- deletion a b e f
4- substitution a b x d e f g
5- insertion a b x c d e f g

Variation in Bacteria

II- Gene transfer

INCLUDES :

1-Transformation

2-Transduction

3-Conjugation

Transformation

Def.:

➢ bacterial cell can take small naked DNA fragments from its surroundings which brings about a genetic change in the recipient cell.

➢ Certain bacteria as(Bacillus, Haemophilus, Neisseria, Pneumococcus) can take up DNA from the environment & can be incorporated into the recipient 'chromosome.

➢ SIGNIFICANCE: INCREASE VIABILITY ,VIRULENCE,

Factors affecting transformation

DNA size state and Competence of the recipient cells

- some bacteria are able to take up DNA naturally only at particular time in their growth cycle when produce ds DNA of at least 5x 10^5 daltons

- Thus, transformation is sensitive to nucleases

Steps in transformation includes:

1- Uptake of DNA

Gram –ve bacteria is taken as dsDNA

Gram +ve bacteria Is taken as ss DNA The complementary is made in the recipient

2 – Gene recombination

After the donor DNA is taken up ,

a reciprocal recombination occurs between the chromosome & the donor DNA

- Recombination requires homology between the DNA& the bacterial recombination genes this called general recombination

transduction

Def.: Transfer of portion of DNA from one bacterial cell to another by bacteriophages .

➢ Bacterial viruses are involved in gene transfer by 3 mechanisms:

1-Generalized 2-abortive

3-specialized

Generalized transduction

➢ Generalized transduction is characterized by the transfer of a generalized group of genes
any gene on the bacterial chromosome have the same chance of being transferred by the viral carrier to another bacterial cell

➢ usually follows a lytic attack of a bacteriophage on bacterial cell, bacterial genome undergoes fragmentation often to a virus sized fragments of DNA

 which can be incorporated inside viral capsids (pseudovirus) can attack a new cell.

 - can be mediated by virulent phages or by temeprate phages

Abortive transduction

➢ In many generalized transduction , the segment of DNA from the donor cell fails to be integrated with the recipient chromosome.

➢ The bacterial DNA remains in the cytoplasm , it does not replicate but transmitted to one daughter cell following each division

Specialized transduction

➢ By which a specific set of genes is transferred by temperate phages from one bacterial cell into another

➢ Temperate phages are those capable of integrating their genetic material into the bacterial chromosome (lysogony)

➢ These phages recombine with the bacterial DNA at precise on the bacterial chromosome

The defective phage :

➢ It misses the viral genes where it left on the bacterial chromosome

➢ Can not replicate itself because of its missing genes

➢ Non defective phage must be present in the same cell (mixed infection) complement the missing segments of the defective virus so that it can be replicated & becomes a transducing phage.

Terms used

1 – Prophage : the viral DNA that integrates into the bacterial chromosome

2 – Temperate phage : Viruses that either lyse the cell or behave as a prophage Can reproduce by 2 mechanisms (the lytic cycle & the lysogenic cycle).

3 – Virulent phage : viruses that only lyse the cell

4- Transducing phage: It is always defective not able to lysis of the infected cell Capable of mobilizing bacterial genetic material from one cell to another .

Plasmid-mediated conjugation (conjugation)

Def of conjugation :

- Is the transfer of DNA from a donor to a recipient by direct physical contact between the cells
- Donor (male) & recipient (female)
- The direction of transfer is one way (donor to recipient)

Mechanism of bacterial conjugation

1- donor cell produce pilus
2 – pilus attaches to recipient cell , brings the 2 cells together
3- the mobile plasmid is nicked & a single strand of DNA is then transferred to the recipient cell
4- both cells recircularize their plasmids, synthesize second strands & reproduce pili, both cells are now viable donors

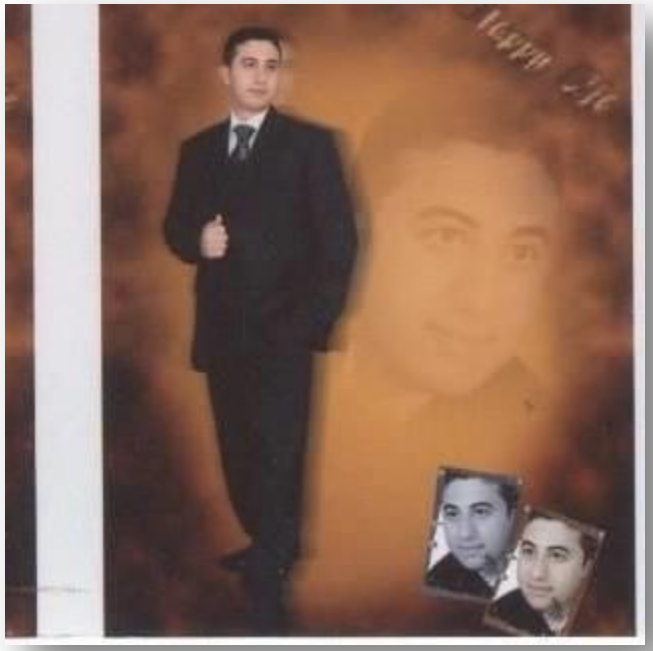
Recombination

- Plasmid DNA can be integrated into bacterial chromosome It is known as episome
- N.B.:

➢ Plasmids of gram positive are non transmissible plasmids as they lack self transfer because they lack genes that code for the sex pili.

Transposons (Jumping genes)

- These are genetic materials that can move (Jump or transposon) from one location to another on the same chromosome or from plasmid to plasmid or from plasmid to chromosome and the reverse
- They can not autonomously replicate but they replicate with the plasmid or chromosome
- Transmissible , Insertion into plasmid can lead to its dissemination



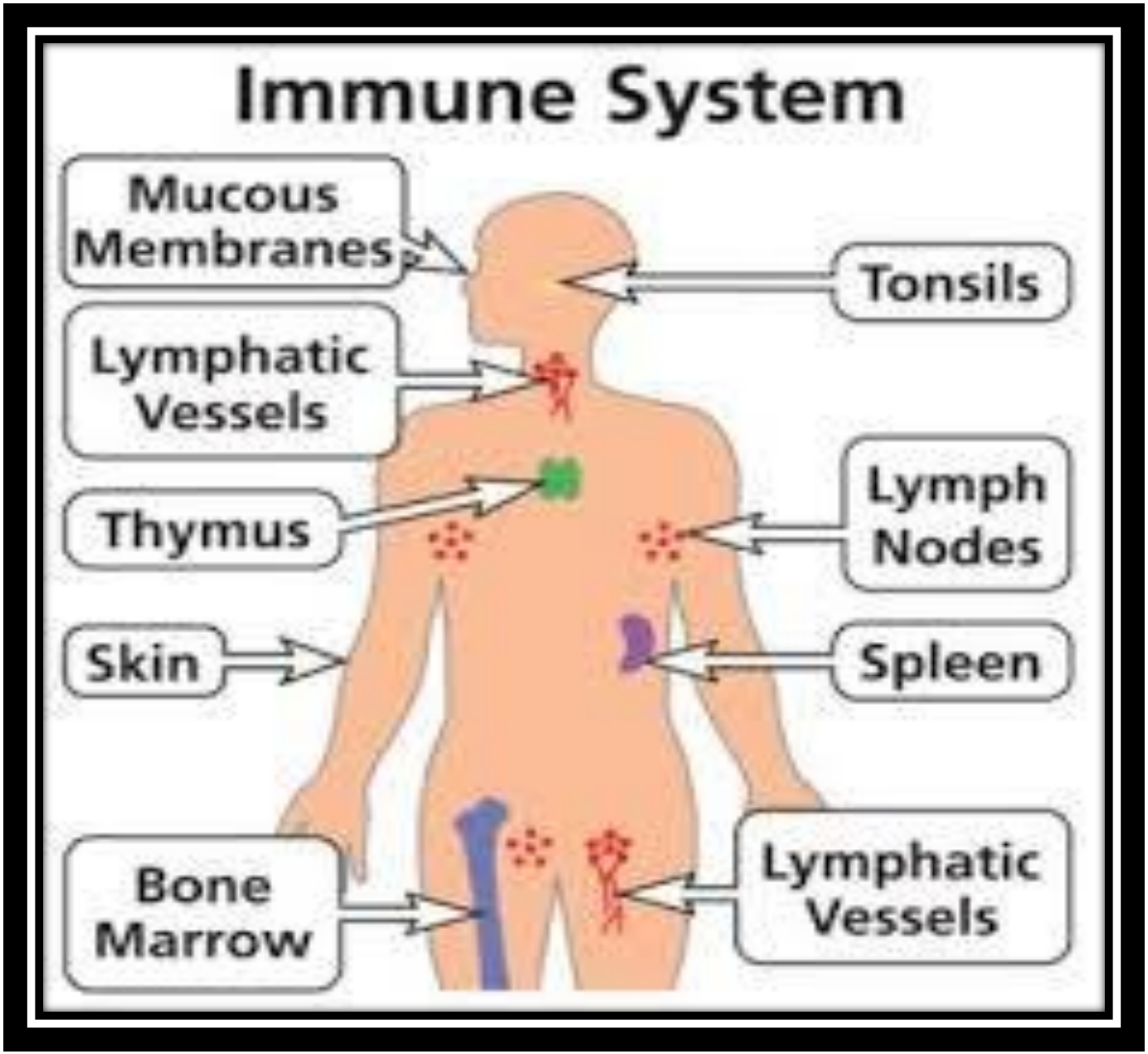
immune system

What is immune system and its function

Your immune system is a large network of organs, white blood cells, proteins (antibodies) and chemicals. This system works together to protect you from foreign invaders (bacteria, viruses, parasites, and fungi) that cause infection, illness and disease.

the organs and processes of the body that provide resistance to infection and toxins. Organs include the thymus, bone marrow, and lymph nodes.

What is the immune system? The immune system protects your child's body from outside invaders. These include germs such as bacteria, viruses, and fungi, and toxins (chemicals made by microbes). The immune system is made up of different organs, cells, and proteins that work together.


Immune System
Mucous Membranes
Tonsils
Lymphatic Vessels
Thymus
Lymph Nodes
Skin
Spleen
Bone Marrow
Lymphatic Vessels

What are the 4 immune systems?

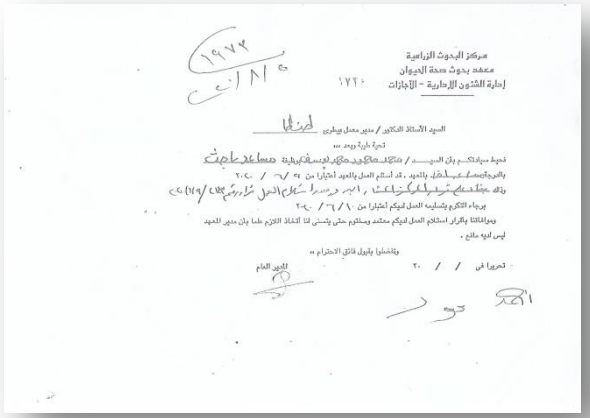
What are the 4 immune systems?
The main parts of the immune system are: white blood cells, antibodies, the complement system, the lymphatic system, the spleen, the thymus, and the bone marrow. These are the parts of your immune system that actively fight infection.

There are 2 main parts of the immune system:

1-The innate immune system. You are born with this.

2-The adaptive immune system. You develop this when your body is exposed to microbes or chemicals released by microbes.

These 2 immune systems work together.



The innate immune system

This is your child's rapid response system. It is the first to respond when it finds an invader. It is made up of the skin, the eye's cornea, and the mucous membrane that lines the respiratory, gastrointestinal, and genitourinary tracts. These all create physical barriers to help protect your child's body. They protect against harmful germs, parasites (such as worms), or cells (such as cancer). The innate immune system is inherited. It is active from the moment your child is born. When this system recognizes an invader, it goes into action right away. The cells of this immune system surround and cover the invader. The invader is killed inside the immune system cells (called phagocytes).

The acquired immune system

The acquired immune system, with help from the innate system, makes special proteins (called antibodies) to protect your body from a specific invader. These antibodies are developed by cells called B lymphocytes after the body has been exposed to the invader. The antibodies stay in your child's body. It can take several days for antibodies to form. But after the first exposure, the immune system will recognize the invader and defend against it. The acquired immune system changes during your child's life. Immunizations train your child's immune system to make antibodies to protect them from harmful diseases.

The cells of both parts of the immune system are made in different organs of the body, including:

Adenoids. Two glands located at the back of the nasal passage.
Bone marrow. The soft, spongy tissue found in bone cavities.
Lymph nodes. Small organs shaped like beans, which are located all over the body and connect via the lymphatic vessels.
Lymphatic vessels. A network of channels all over the body that carries lymphocytes to the lymphoid organs and bloodstream.
Peyer patches. Lymphoid tissue in the small intestine.
Spleen. A fist-sized organ located in the belly (abdominal) cavity.
Thymus. Two lobes that join in front of the windpipe (trachea) behind the breastbone.
Tonsils. Two oval masses in the back of the throat.

THE TOPIC TODAY

How do vaccines work?

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

Germs are all around us, both in our environment and in our bodies. When a person is susceptible and they encounter a harmful organism, it can lead to disease and death.
The body has many ways of defending itself against pathogens (disease-causing organisms). Skin, mucus, and cilia (microscopic hairs that move debris away from the lungs) all work as physical barriers to prevent pathogens from entering the body in the first place.
When a pathogen does infect the body, our body's defences, called the immune system, are triggered and the pathogen is attacked and destroyed or overcome.

The body's natural response

A pathogen is a bacterium, virus, parasite or fungus that can cause disease within the body. Each pathogen is made up of several subparts, usually unique to that specific pathogen and the disease it causes.

The subpart of a pathogen that causes the formation of antibodies is called an antigen.

The antibodies produced in response to the pathogen’s antigen are an important part of the immune system.

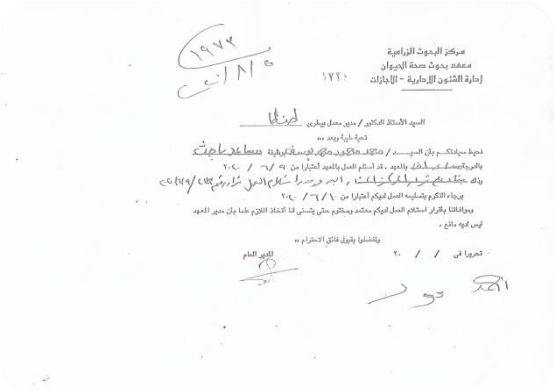
You can consider antibodies as the soldiers in your body's defense system.

Each antibody, or soldier, in our system is trained to recognize one specific antigen.

We have thousands of different antibodies in our bodies.

When the human body is exposed to an antigen for the first time, it takes time for the immune system to respond and produce antibodies specific to that antigen.

In the meantime, the person is susceptible to becoming ill.



مركز البحوث الزراعية
معهد بحوث صحة الحيوان
إدارة الشئون الإدارية - الإجازات

السيد الأستاذ الدكتور / مدير معمل بيطري

تحية طيبة وبعد ،،

تحيط سيادتكم بأن السيد / [illegible] بوسطة بقرية [illegible] مطاعجة
والمرفق بصحبته عبارة عن [illegible] بالمعهد ، قد استلم العمل بالمعهد اعتبارا من ٢٠٢٠/٦/٩
بناء عليه نرجو [illegible] ، [illegible] ١٩٧٢/٥٢ بتاريخ ٢٠٢٠/٦/٩

يرجاء التكرم بتأشيره العمل ليكم اعتبارا من ٢٠٢٠/٦/١٠
وهذا بالإقرار استلم العمل ليكم معتمد وبختوم حتى يتسنى لنا اتخاذ اللازم علما وان مدير المعهد
ليس فيه مانع .

وتفضلوا بقبول فائق الاحترام ،،

تحريرا في / / ٢٠

المدير العام

جامعة بنها

شهادة مؤقتة

تشهد كلية الطب البيطري

منح السيدة : محمد محمود محمد يوسف

المولودة في : البحيرة بتاريخ : ٢٩/٩/١٩٩٢

الجنسية : مصرى الرقم القومي : ٢٩٢٠٩٠٩١٨٠٠٢٦

درجة الماجستير في العلوم الطبية البيطرية تخصص:

"البكتريولوجيا والمناعة والفطريات"

وموضوع الرسالة: تواجد ميكروبات التسمم الغذائي في بعض أنواع اللحوم الطازجة في السوق المحلي.

تاريخ اعتماد مجلس الكلية : ٢٠٢١/٤/٢٧

تاريخ اعتماد مجلس الجامعة : ٢٠٢١/٥/١٩

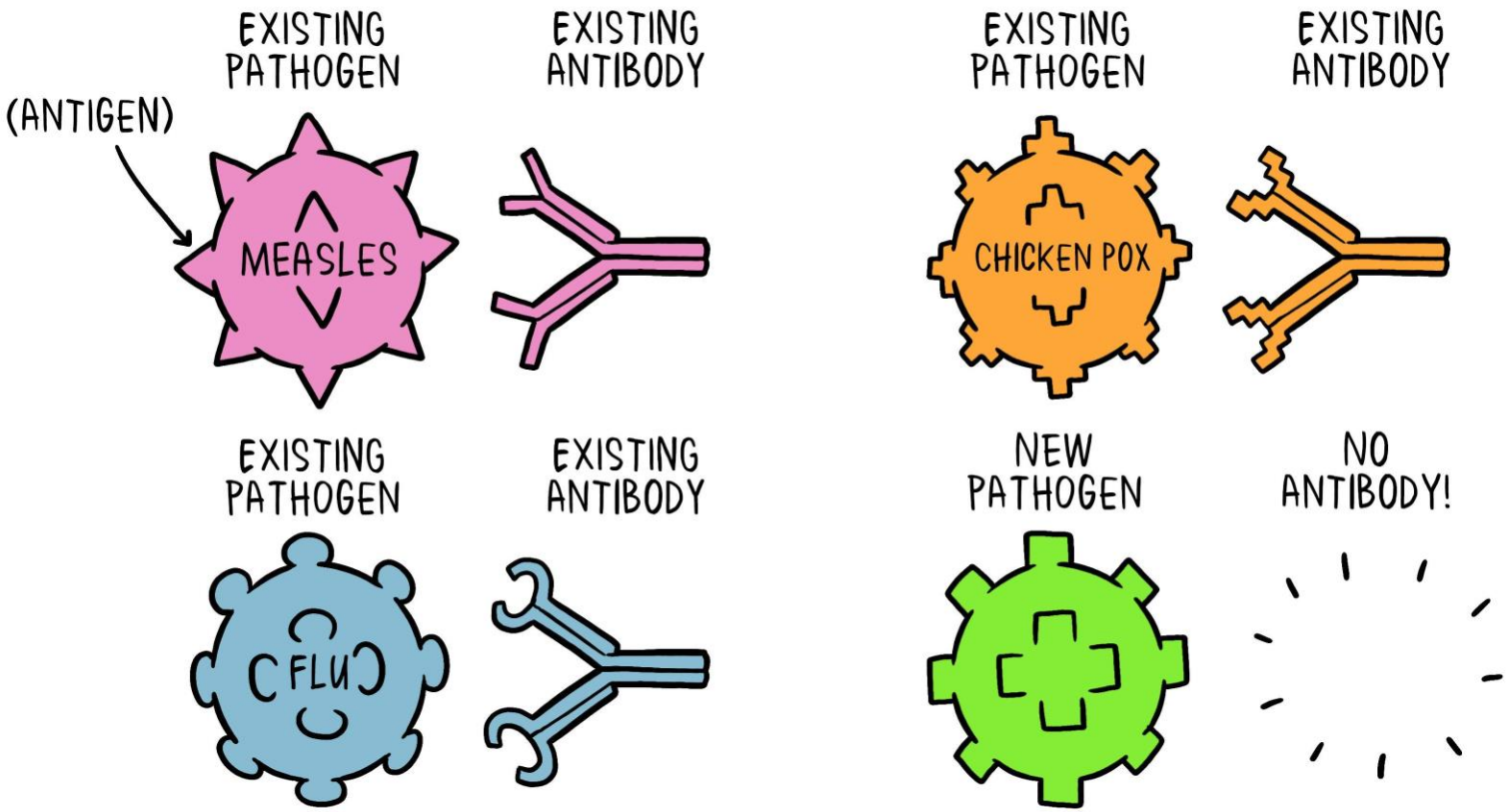
Once the antigen-specific antibodies are produced, they work with the rest of the immune system to destroy the pathogen and stop the disease.

Antibodies to one pathogen generally don't protect against another pathogen except when two pathogens are very similar to each other, like cousins.

Once the body produces antibodies in its primary response to an antigen, it also creates antibody-producing memory cells, which remain alive even after the pathogen is defeated by the antibodies.

If the body is exposed to the same pathogen more than once, the antibody response is much faster and more effective than the first time around because the memory cells are at the ready to pump out antibodies against that antigen.

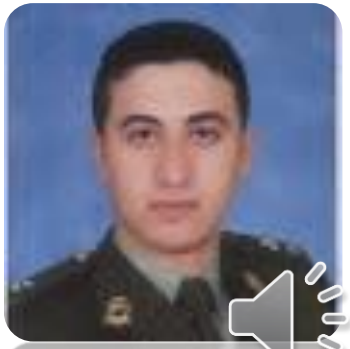
This means that if the person is exposed to the dangerous pathogen in the future, their immune system will be able to respond immediately, protecting against disease.



When a new pathogen or disease enters our body, it introduces a new antigen. For every new antigen, our body needs to build a specific antibody that can grab onto the antigen and defeat the pathogen.

How vaccines help

Vaccines contain weakened or inactive parts of a particular organism (antigen) that triggers an immune response within the body.



Newer vaccines contain the blueprint for producing antigens rather than the antigen itself.

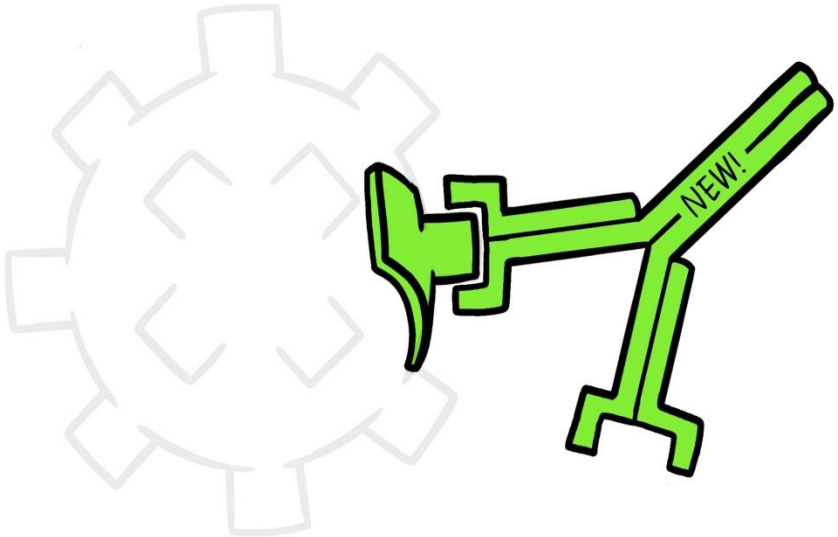
Regardless of whether the vaccine is made up of the antigen itself or the blueprint so that the body will produce the antigen, this weakened version will not cause the disease in the person receiving the vaccine,

but it will prompt their immune system to respond much as it would have on its first reaction to the actual pathogen.

.MOHAMMED

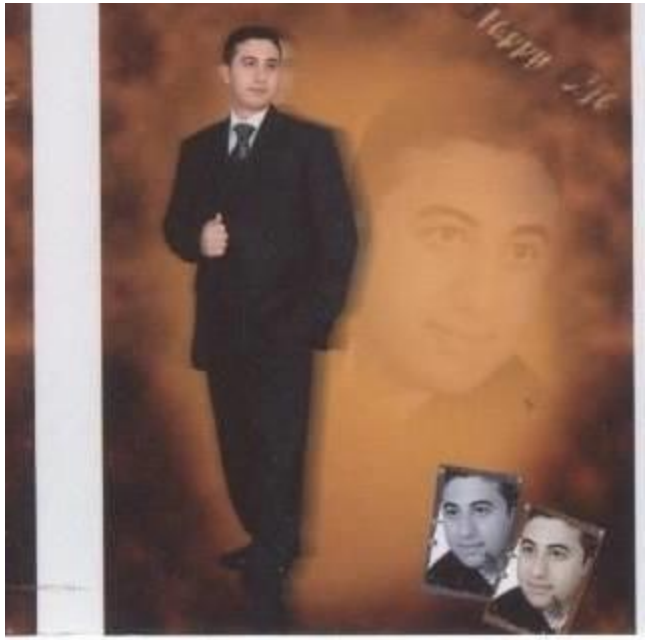
VACCINE

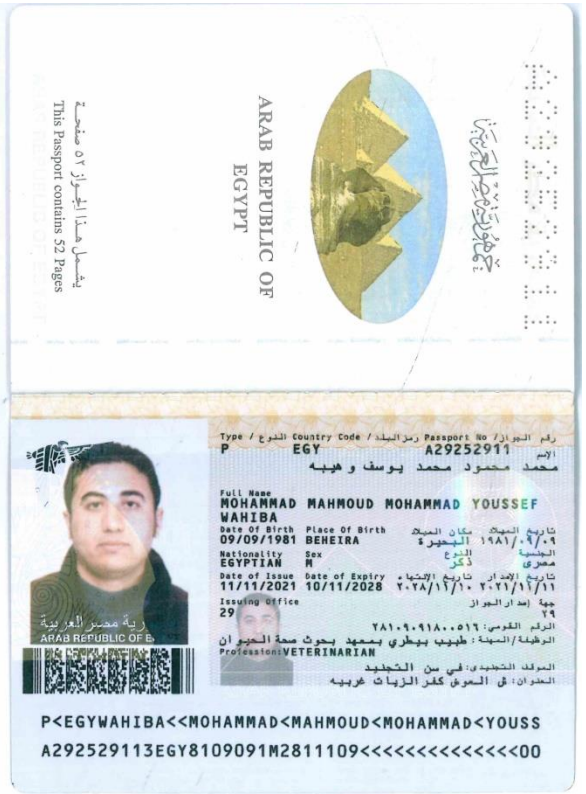
NEW ANTIBODY



A VACCINE is a tiny weakened non-dangerous fragment of the organism and includes parts of the antigen. It's enough that our body can learn to build the specific antibody. Then if the body encounters the real antigen later, as part of the real organism, it already knows how to defeat it.

Some vaccines require multiple doses, given weeks or months apart. This is sometimes needed to allow for the production of long-lived antibodies and development of memory cells. In this way, the body is trained to fight the specific disease-causing organism, building up memory of the pathogen so as to rapidly fight it if and when exposed in the future.





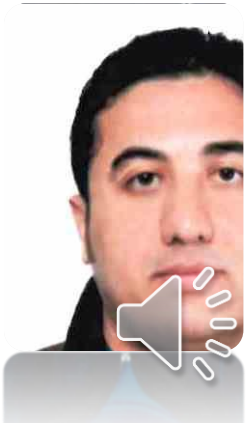
Herd immunity

When someone is vaccinated, they are very likely to be protected against the targeted disease.

But not everyone can be vaccinated. People with underlying health conditions that weaken their immune systems (such as cancer or HIV) or who have severe allergies to some vaccine components may not be able to get vaccinated with certain vaccines.

These people can still be protected if they live in and amongst others who are vaccinated.

When a lot of people in a community are vaccinated the pathogen has a hard time circulating because most of the people it encounters are immune.

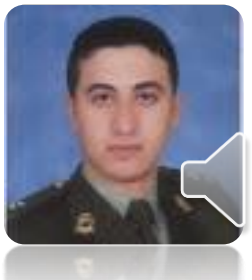


COMMUNITY VACCINATION PROTECTS THE WHOLE COMMUNITY EVEN THOSE WHO CAN NOT VACCINATED

This is called herd immunity.

Throughout history, humans have successfully developed vaccines for a number of life-threatening diseases, including meningitis, tetanus, measles and wild poliovirus.

In the early 1900s, polio was a worldwide disease, paralysing hundreds of thousands of people every year. By 1950, two effective vaccines against the disease had been developed. But vaccination in some parts of the world was still not common enough to stop the spread of polio, particularly in Africa. In the 1980s, a united worldwide effort to eradicate polio from the planet began.



Over many years and several decades, polio vaccination, using routine immunization visits and mass vaccination campaigns, has taken place in all continents. Millions of people, mostly children, have been vaccinated and in August 2020, the African continent was certified wild poliovirus free, joining all other parts of the world except Pakistan and Afghanistan, where polio has not yet been eradicated.

(SPONDYLOSIS)

(داء الفقار) (خشونة الفقرات)

مساعدباحث /محمدمحمودمحمديوسف وهيبه

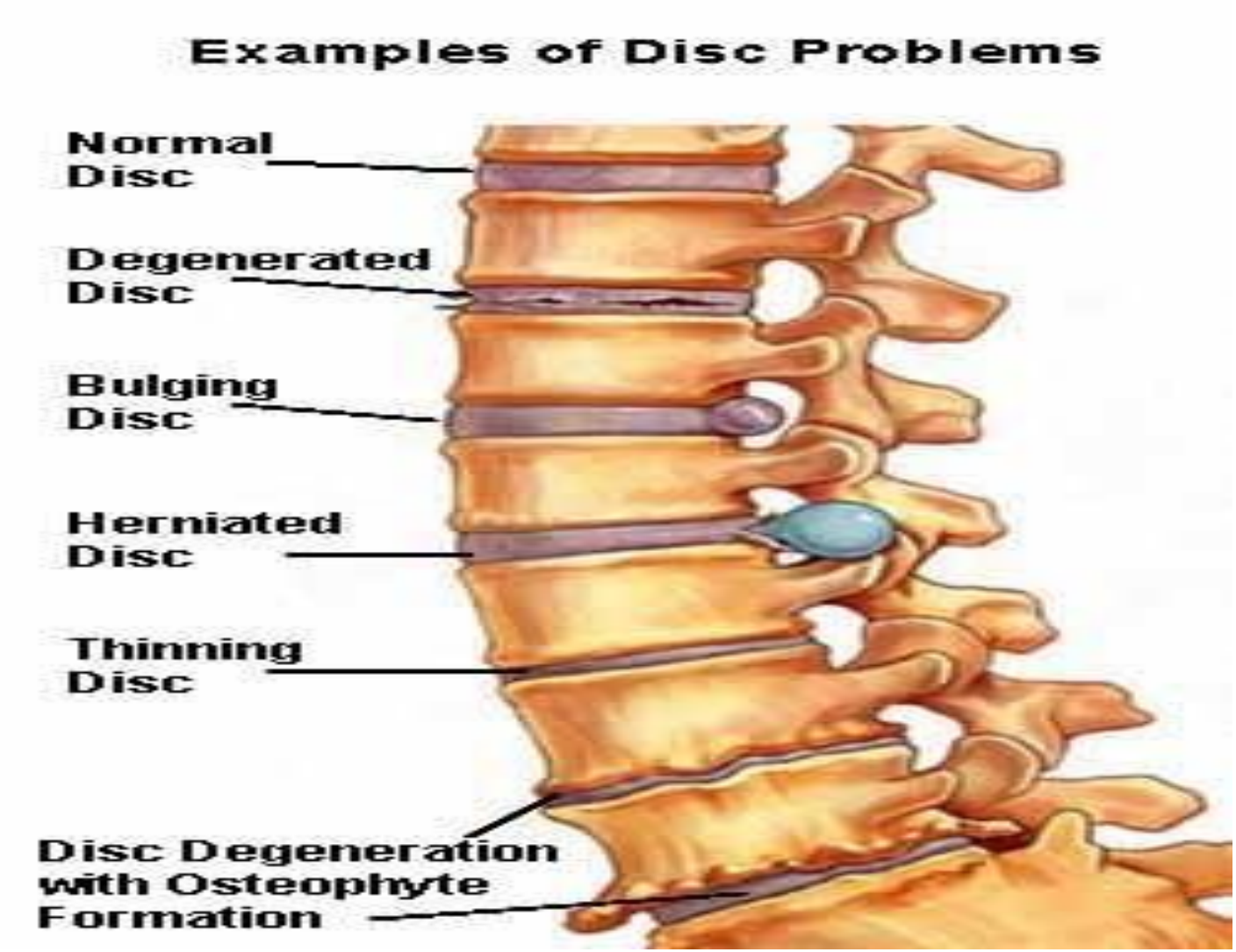
معهدبحوث الصحة الحيوانية

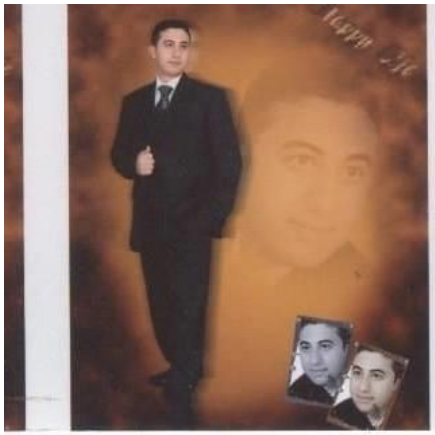
مركزالبحوث الزراعية

وزارة الزراعة

جمهورية مصرالعربية

يُعد داء الفقار الرقبية (خشونة الفقرات) مصطلحًا عامًا يصف التلف والتمزق المرتبط بالمرحلة العمرية والذي يصيب أقراص العمود الفقري في الرقبة.
وتظهر علامات الفُصال العظمي كلما أصاب الجفاف والانكماش الأقراص، بما يتضمن النتوءات العظمية على طول أطراف العظام. ويُعد داء الفقار الرقبية منتشرًا للغاية ويتفاقم مع التقدم في العمر.

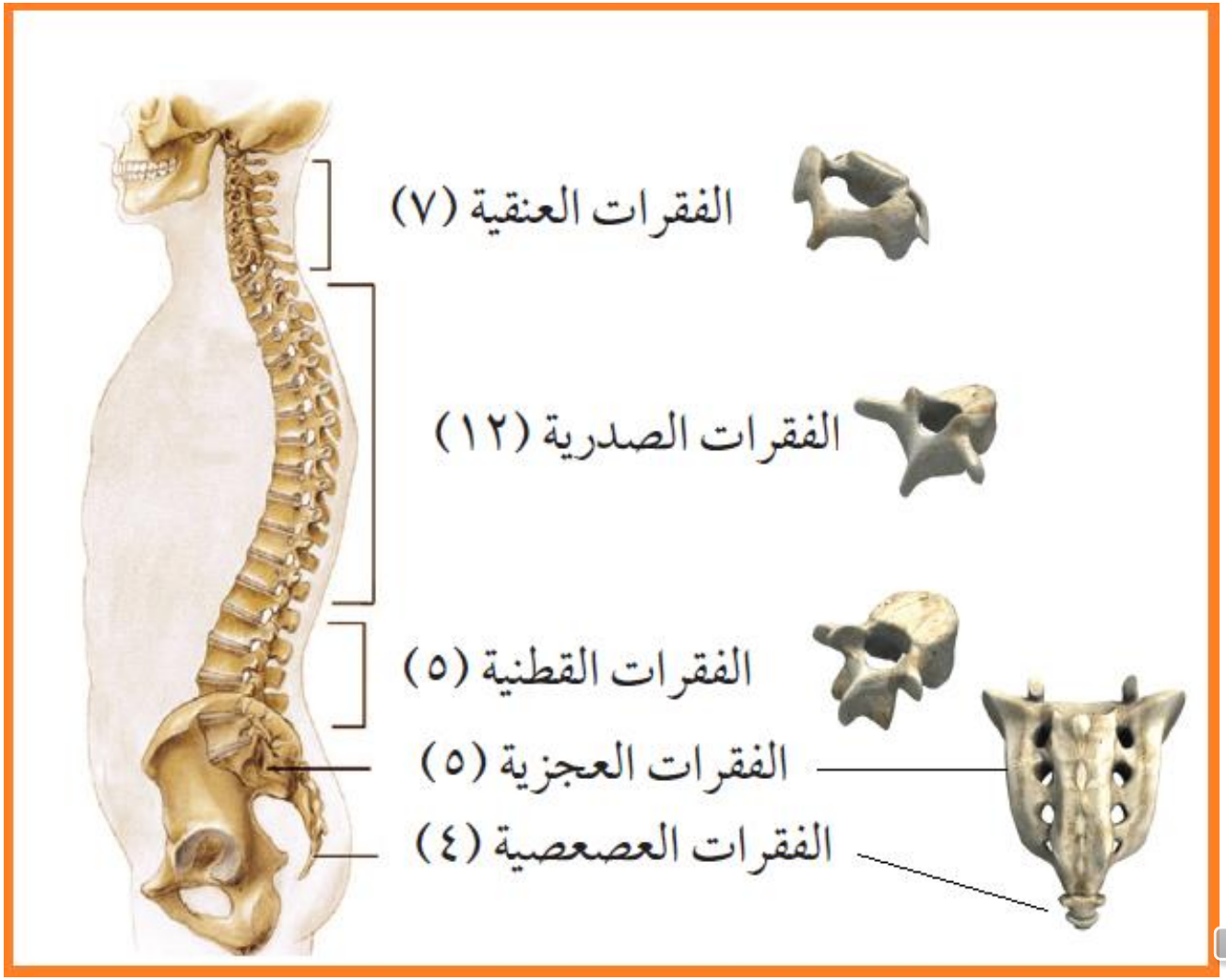

Examples of Disc Problems
Normal Disc
Degenerated Disc
Bulging Disc
Herniated Disc
Thinning Disc
Disc Degeneration with Osteophyte Formation



الفصال العظمى

ويُعرَف الالتهاب المفصلي العظمي في أغلب الحالات بالتآكل والاهتراء الناجمين عن استخدام المفصل بمرور الزمن. ولا يستهدف الالتهاب المفصلي العظمي النسيج الغضروفي وحسب، بل يؤثر كذلك على المفصل بالكامل. ويسبب تغيرات في العظام وتآكل الأنسجة الضامة التي تربط المفصل معًا وتربط العضلات بالعظام.

العمود الفِقْرِي أو العمود الفَقْرِي أو الفَقَار للكائنات الحية الفقارية مكون من سلسلة فِقْرَات مفرغة لحفظ النخاع الشوكي الذي يمتد فيها ويبلغ عدد الفِقْرَات عند الإنسان ٣٣ فقرة. يدعى الفراغ الذي يسكنه النخاع الشوكي بالنفق الفقري.


الفقرات العنقية (٧)
الفقرات الصدرية (١٢)
الفقرات القطنية (٥)
الفقرات العجزية (٥)
الفقرات العصعصية (٤)

لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة. اليكم لمحة تشريحية عن العمود الفقري

يتصل العمود الفقري في الأعلى مع الجمجمة وفي الأسفل مع الحوض، وهو يتكون من ٢٤ فقرة متحركة إضافة إلى ٧ فقرات أخرى ملتحمة تشكل القسم السفلي من العمود الفقري (العجز والعصعص).

يقسم العمود الفقري إلى المناطق التالية:

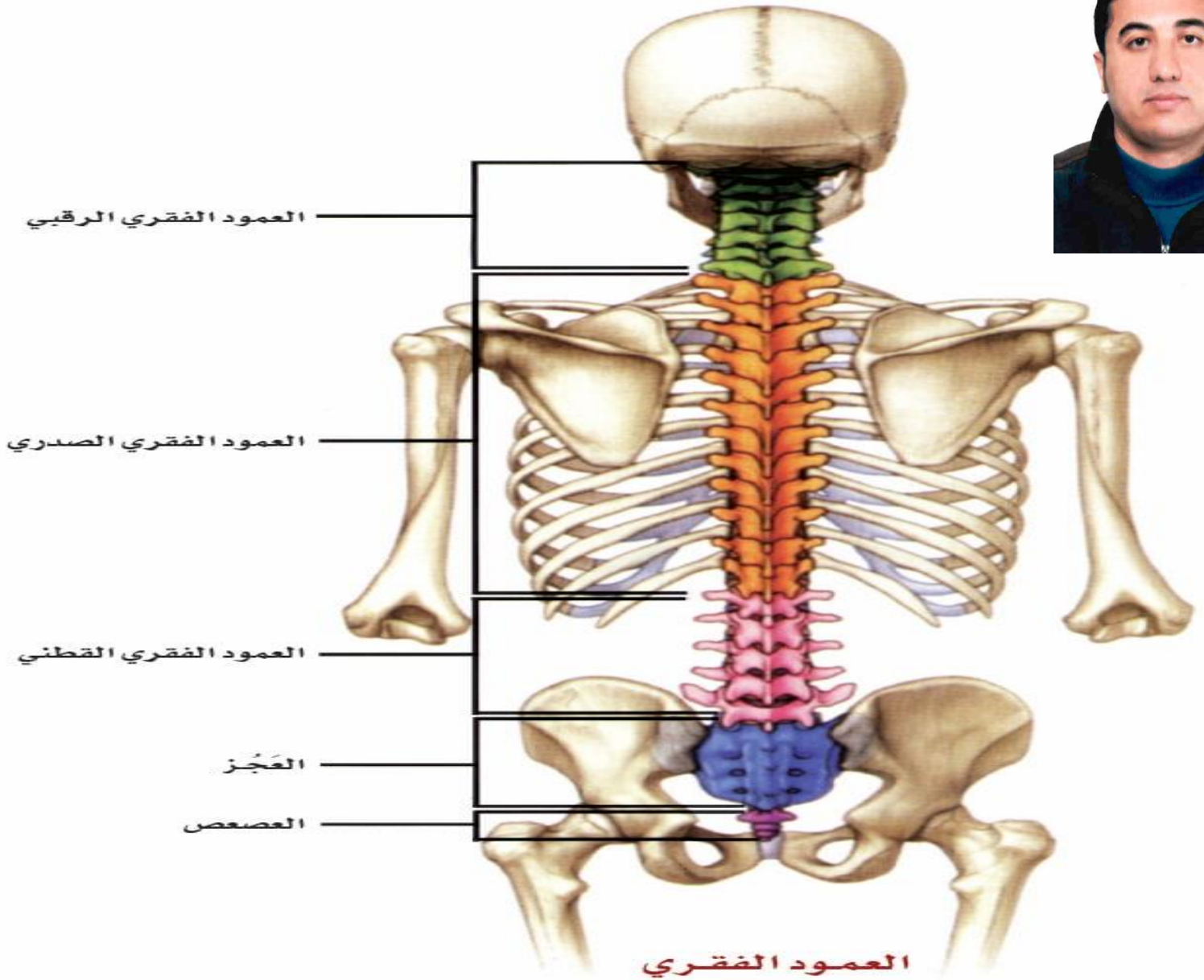
١. الفقرات الرقبية وعددها ٧ فقرات تشكل الانحناء الرقبي للعمود الفقري حيث تنحني هذه الفقرات بشكل طبيعي للأمام.

٢. الفقرات الظهرية أو العمود الفقري الصدري وعددها ١٢ فقرة ويكون انحناء العمود الفقري في هذه المنطقة نحو الخلف، وتتصل الفقرات الظهرية مع الأضلاع التي تكوّن القفص الصدري.

٣. الفقرات القطنية وعددها ٥ فقرات وهي تشكل منطقة أسفل الظهر ويكون انحناء العمود الفقري في هذه المنطقة للأمام بنفس جهة انحناء العمود الرقبي.

٤. العجز المكون من ٥ فقرات ملتحمة مع بعضها مشكلة قطعة عظمية واحدة مثلثية الشكل تقع تحت العمود الفقري القطني مباشرة.

٥. العصعص ويتكون من ٤ أو ٥ فقرات ملتحمة وهو القسم الانتهائي من العمود الفقري.



العمود الفقري

بيانات هامة للإتصال بصاحب

الموبايل نوكيا 130 –

دكتورمحمدمحمودوهيبه وعائلته :

دكتورمساعدباحث

/محمدمحمودمحمديوسف وهيبه

01210973074

أبى 01221868424

أمى 01206372319

أختى 01220781971

أخى 01288849405

دكتور عادل عبدالعزيز مدير العمل

01006646670

جامعة سوهاج
كلية الطب البيطري
لضمان جودة التعليم والاعتماد
لتدعيم الطلاب

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١ م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس في العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جداً) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

بيان التقديرات الحامل عليها الثناء، مراعاته بالكلية :-

الفرقة الأولى: دور: مايو عام: ١٩٩٨/١٩٩٩م

المادة	الفيزياء: العيوبة	الميكانيكا: ميكانيكا وصوتيات	الكيمياء: العامة	التشريح والأنسجة	المستويات	الوراثة والخدمة	أحياء: عيوب	لغة إنجليزية	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	ممتاز	جيد//	ممتاز	جيد جداً
الدرجة	٣٥	٧٦	٨٢	٧٧	٧٤	٩٤	٢٣	٤٥	٥١٦

الفرقة الثانية: دور: مايو عام ١٩٩٩/٢٠٠٠م

المادة	تشريح الطيور ومكارن	الفسيولوجيا	طفيليات: عمومية	تغذية الحيوان والدواجن وأمراض سوء التغذية	سلوكيات الحيوان والدواجن وبر دواجنها	خاص الفم	التقدير العام
التقدير	جيد جداً	جيد جداً	ممتاز	جيد//	مقبول	مقبول	جيد جداً
الدرجة	٨١	٨٢	٩١	٧٢	٦٤	٣٠	٣٨٩

الفرقة الثالثة: دور: مايو عام: ٢٠٠٠/٢٠٠١م

المادة	باثولوجيا عام	مختبرباثولوجيا وطفيليات البعامة	التطفيليات	فارماكولوجيا	إنعاش حيواني	القضاء وإدارة برام م	فيروليوجيا	التقدير العام
التقدير	مقبول	جيد جداً	جيد جداً	مقبول	جيد//	جيد//	مقبول	جيد//
الدرجة	٥٠	٨١	٧٨	٧١	٦٩	٧٤	٣٢	٤٥٥

الفرقة الرابعة: دور: مايو عام: ٢٠٠١/٢٠٠٢م

المادة	باثولوجيا تشريحيه مرضي	أمراض باطنة	جراحة عامة (تناسلية)	جراحة عامة وتخدير	باثولوجيا وطب شرعي	الرقابة الصحية على الألبان	باثولوجيا إكلينيكية	أمراض التناسلية	التقدير العام
التقدير	جيد//	جيد جداً	جيد جداً	جيد جداً	جيد//	جيد//	جيد//	جيد جداً	جيد//
الدرجة	٧٤	٦٩	٧٧	٧٦	٧٠	٧٣	٣٥	٤٠	٥١٤

الفرقة الخامسة: دور: مايو عام ٢٠٠٢/٢٠٠٣م

المادة	أمراض باطنه	جراحة عامة والتخدير	التوليد والتلقيح الصناعي	أمراض تناسلية	أمراض الطيور والأرانب	الرقابة الصحية على اللحوم ومنتجاتها	صحة الحيوان والدواجن والبيئة	الأمراض المشتركة	التقدير العام
التقدير	مقبول	جيد جداً	جيد//	جيد جداً	جيد//	جيد//	جيد//	ممتاز	جيد//
الدرجة	٦٤	٧٩	٦٩	٨٢	٧٣	٦٧	٦٦	٨٥	٥٨٥

وقد أعطيت له هذه الشهادة بناء على طلبه وتحت مسئوليته لتقديمها إلى من يهمه الأمر

تحريراً في: ٢٠١٨/١٠/٣٠م

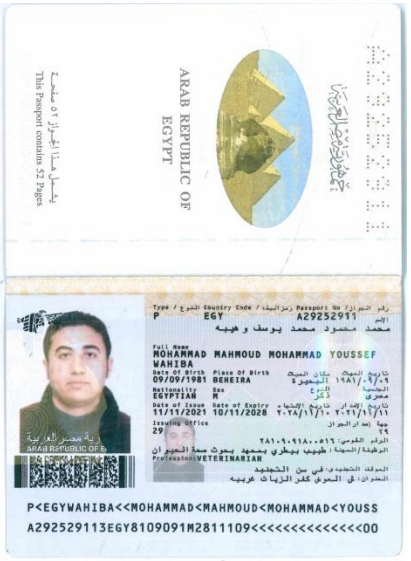
الموظف المختص: أسماء

محمل الطلبة: [illegible]

وكيل الكلية لشئون التعليم والطلاب

عميد الكلية

((أ.د/ محمود عبد الله [illegible])) ((أ.د/ إسماعيل إسماعيل إبراهيم))



يحتوي هذا الجواز ٥٢ صفحة
This Passport contains 52 Pages

ARAB REPUBLIC OF EGYPT

جمهورية مصر العربية

Type / النوع P | Country Code / رمز البلد EGY | Passport No / رقم الجواز A02925911

الإسم محمد محمود محمد يوسف وهيبه

Full Name MOHAMMAD MAHMOUD MOHAMMAD YOUSSEF WAHIBA

Date of Birth 09/09/1981 تاريخ الميلاد ٠٩/٠٩/١٩٨١

Place Of Birth BEHEIRA مكان الميلاد البحيرة

Nationality EGYPTIAN الجنسية مصري

Sex M النوع ذكر

Date of Issue 11/11/2021 تاريخ الإصدار ١١/١١/٢٠٢١

Date of Expiry 10/11/2028 تاريخ الإنتهاء ١٠/١١/٢٠٢٨

Issuing Office جهة إصدار الجواز 29

الرقم القومي: ٢٨١٠٩٠٩١٤٠٠٩١٦

الوظيفة/المهنة: طبيب بيطري

Profession: VETERINARIAN

العنوان: في المنوفية ق: كفر الزيات عربيه

الموقف التجنيد: في سن التجنيد

ARAB REPUBLIC OF EGYPT

P<EGYWAHIBA<<MOHAMMAD<MAHMOUD<MOHAMMAD<YOUSS

A02925911 3EGY8109091M2811109<<<<<<<<<<<<<<00

تتكون كل فقرة من جسم الفقرة وهو القسم الأمامي منها ومن بروزات عظمية عديدة ندعوها النواتئ ترتكز العضلات عليها، ويوجد في مركز كل فقرة حفرة محمية بالعظم المحيط بها، وتشكل الحفر مع بعضها قناة ضمن العمود الفقري هي القناة الشوكية التي يسكنها النخاع الشوكي.

يوجد بين كل فقرتين وسادة اسفنجية اسطوانية الشكل ندعوها القرص بين الفقرات (أو الديسك)، ويتكون هذا القرص من نواة جيلاتينية طرية ندعوها النواة اللبية تتوضع في مركز القرص، ويحيط بها حلقة ليفية قوية مكونة من شرائح متوضعة بشكل متحد المركز تماماً مثل شرائح البصل، ويعتبر القرص بين الفقرات مفصلاً يسمح بحركة العمود الفقري. وهناك ٢٣ قرصاً بين الفقرات يشكلون ثلث ارتفاع العمود الفقري ويخضعون لعمل مستمر، فهذه الأقراص هي التي تمتص الصدمات وتغيرات الضغط التي يخضع لها العمود الفقري، وتكون وظيفة الفقرات الصلبة تحويل هذه الضغوط إلى الأقراص بين الفقرات الملزمة بالتكيف مع الضغوط المختلفة، وفي الحقيقة يؤدي تعرض القرص إلى ضغط شديد إلى تسطح النواة اللبية بغية توزيع الضغط على أكبر مساحة ممكنة، وهذا يؤدي إلى تمدد الحلقة الليفية. وأثناء الراحة يحدث العكس حيث تشتد ألياف الحلقة الليفية ويستعيد القرص ارتفاعه الطبيعي.

بيانات ومعلومات طالب الزواج

بيانات شخصية :

- الإسم/ محمدمحمودمحمديوسف وهيبه
- العمل /مساعدباحث بمعهد بحوث الصحة الحيوانية
- المؤهل / ماجستير البكتيريا والمناعة والفطريات
- (عام 2021/5/19)
- العمر / أربعون عاما
- الحالة الإجتماعية / أعزب لم يسبق لى الإرتباط والزواج
- الخدمة العسكرية / تمت تأديتها
- الإقامة / شارع مجمع المدارس/البيت الأبيض المقابل لمحل ماكس جيم لكمال الأجسام/الدور الثانى ملكى/منزل الأستاذ محمودوهيبه /منطقة العوض/قرية الدلجمون /مركز كفرالزيات / محافظة الغربية
- رقم الموبايل / 01210973074
- البريد الالكتروني : m0hammed_yousef@yahoo.com
- الطول 180سم
- الوزن 100كيلوجرام
- لون البشرة أبيض
- الراتب حوالى 5000 جنيه شهريا
- المطلوب عروسة وزوجة مناسبة لى

بيانات وظيفية :

التعيين / بمعهد بحوث الصحة الحيوانية بالدقي / مصر/فرع طنطا كالتالي :-

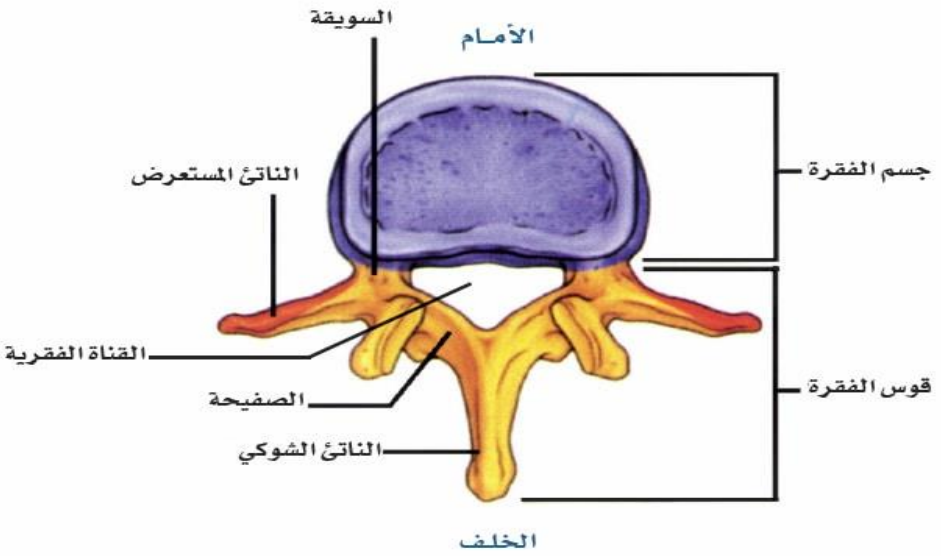
1- طبيب بيطري (من عام 2009إلي 2020)
2- مساعد باحث (من عام 2020/6 الي الوقت الجاري)
3- الحصول على درجة الماجستيرفى البكتيريا والمناعة والفطريات (بتاريخ 2021/5/19)
4- طبيب بيطرى فى الخدمة العسكرية الإلزامية فى الرقابة الصحية على الأغذية

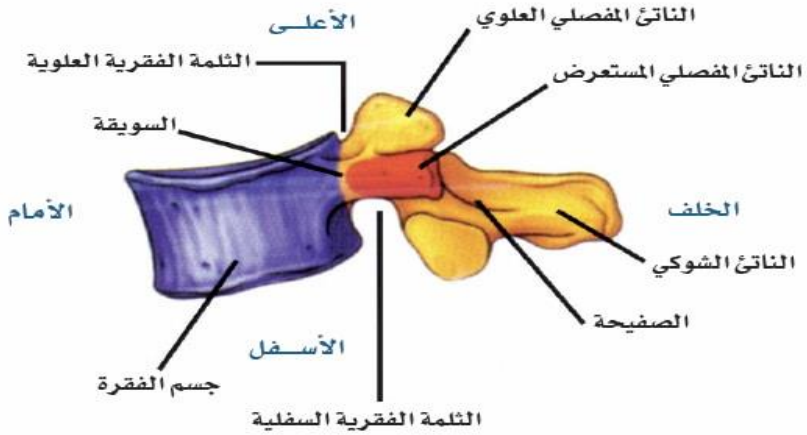
(اللحوم-الألبان ومنتجاتها)

المطلوب من معاليكم زوجة مناسبة

ولسيادتكم وافر التحية والتقديروالإحترام

متمنيا لسعادتكم مزيدا من التقدم والرقي





داء الفقار

دَاء الفَقَار أو تَنَكُّس الفَقار هو انتكاس العمود الفقري لأي سبب. بالمعنى الدقيق، يشير إلى هشاشة العظام في العمود الفقري، وهو تآكل وتمزق العمود الفقري المرتبط بتقدم العمر، وهو السبب الأكثر شيوعًا للإصابة بداء الفقار. تؤثر العملية التنكسية في هشاشة العظام بشكل رئيسي على الأجسام الفقرية، والثقب العصبي، ومفاصل الوجه (متلازمة الوجه). إذا كانت شديدة، فقد تسبب ضغطًا على النخاع الشوكي أو جذور الأعصاب مع اضطرابات حسية أو حركية لاحقة، مثل الألم والتنمل وعدم التوازن وضعف العضلات في الأطراف

عندما تقل المسافة بين فقرتين متجاورتين، قد يؤدي ضغط جذر العصب الخارج من الحبل الشوكي إلى اعتلال الجذور (اضطرابات حسية وحركية، مثل ألم شديد في الرقبة أو الكتف أو الذراع أو الظهر أو الساق، مصحوبًا بضعف العضلات).

أقل شيوعًا، قد يؤدي الضغط المباشر على النخاع الشوكي (عادةً في العمود الفقري العنقي) إلى اعتلال النخاع، الذي يتميز بضعف عالمي، وخلل في المشي، وفقدان التوازن، وفقدان السيطرة على الأمعاء أو المثانة. قد يعاني المريض من صدمات (تنمل) في اليدين والرجلين بسبب انضغاط العصب وقلة تدفق الدم. في حالة إصابة فقرات العنق، يتم تسميتها بداء الفقار الرقبية. يسمى داء الفقار أسفل الظهر داء الفقار القطني. المصطلح مشتق من اليونانية القديمة σπόνδυλος *spóndylos*، «فقرات - »، في الجمع «فقرة»، العمود الفقري».

جمهورية مصر العربية

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية **الطـــب البيـــطري**

أن / **محمـــد محمـــود محمـــد يوسف**

المولود فى **البحيرة** بتاريخ **١٩٨١م**

قد حصل على درجة **البكالوريوس فى العلوم الطبية البيطرية**

فى دور **مايو** عام **٢٠٠٣م** بتقدير **جيـــد جـداً**

مجموع تراكمى **٢٤٧٥ / ٣٣٠٠** النسبة المئوية **٧٥,٠٠%**

و قد اعتمد مجلس الكلية نتيجة إمتحانه فى **٢٠٠٣/٧/١٥م**

ومجلس الجامعة فى **٢٩ /٢٠٠٣/٧م** **بمقتضى قرار مجلس جامعة طنطا**

الموظف المسئول | المراجع | مدير شئون الطلاب | عميد الكلية

تحريراً فى **٢٠١٢/٥/٢٢م**

جمهورية مصر العربية

وزارة الداخلية

قطاع مصلحة الأحوال المدنية

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صورة قيد الميلاد

رقم القومى ٣٨١٠٩٠٩ ١٨ ٠٥١٦ ٠

بيانات المولود

محمد

الجنسيه :مصر

الديانه :مسلم

النوع :ذكر

محل الميلاد :البحيرة / مركز ابوحمص

تاريخ الميلاد :تسعة من سبتمبر

عام الف و تسعمائه و واحد و ثمانون

بيانات الأب

محمود محمد يوسف وهيبه

الديانه :مسلم

الجنسيه :مصر

بيانات الأم

ليلى محمد عبد المنعم

الديانه :مسلمة

الجنسيه :مصر

م . صحة : ابوحمص المختار رقم القيد :١٧٨٣

س .مدني : مركز ابوحمص ت .القيد :٣٠ /١٠ /١٩٨١

س . امد آر : سجل كفر الزيات ت .امد آر :٢٨ /٠٩ /٢٠١٧

رقم مسلسل ٢١٩٠٤٧٨٥٤٠

توقيع

تأكد من وجود طابع الأمومة والطفولة هلة ، جنيه والعلامة المائية ، نسر شعار الجمهورية ، وشهادة أحوال مدنية.

POLICE PRESS /2590630

((نموذج رقم ٢٥ س))

٣٥٤٣٣٧٣

إصدار ثانى

وزارة الدفاع
شهادة تأدية الخدمة العسكرية
بالقوات المسلحة بجمهورية مصر العربية

تشهد وزارة الدفاع

أن

رقم عسكرى: ٢٠٠٤٥٧٨٨٠١٩٦٦

درجة: ملازم مجند — اسم: محمد محمود محمد يوسف

الرقم القومى: ٢٨١٠٩٠٩١٨٠٠٥١٦ — سجل مدنى: مغر الزيات

محافظة: الغربية

قد انتهت مدة خدمته العسكرية
قوات الجيش
عن مدة خدمة حسنة قدرها

يوم ٥ شهر ١ سنة

بتاريخ يوم ٠١ شهر ٠٩ سنة ٢٠٠٥

بسبب نقله الى الاحتياط تنقضى مدة خدمته بالقوات الاحتياطية

يوم شهر سنة

وكانت درجة أخلاقه أثناء الخدمة قدوة حسنة

تحريرا بتاريخ ٢٠١٩/٠١/٢١

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

بيان مدة الخدمة العسكرية/ كود التعبئة ٥٩٠١٠١٠٠٣

البيان	يوم	شهر	سنة
تاريخ الالتحاق	٢٦	٠٧	٢٠٠٤
تاريخ انهاء الخدمة	٠١	٠٩	٢٠٠٥
اجمالي مدة الخدمة	٥	١	١
المدة الفائدة	٠	٠	٠
صافي مدة الخدمة	٥	١	١

من			إلى			المدة		
يوم	شهر	سنة	يوم	شهر	سنة	يوم	شهر	سنة

الموقف من الخدمة بالقوات الاحتياطية

يتم الرجوع لإدارة شئون ضباط القوات المسلحة لحين بلوغ سن التقاعد للرتبة

تعليمات ونصائح

١ - حافظ على هذه الشهادة سليمة وقدمها إلى كل جهة تطلبها منك.

٢ - الخدمة بالقوات الاحتياطية جزء من الخدمة العسكرية المفروضة عليك قانونا ويجب تأديتها كاملة.

٣ - عندما تستدعى للخدمة بالقوات الاحتياطية عليك التقدم إلى الجهة أو الوحدة التى ستحددها لك تعليمات الاستعداد فى التاريخ الذى يعين لذلك.

٤ - التخلف عن الحضور يوقعك تحت طائلة العقوبة التى قررها القانون لذلك.

٥ - اذا ما حدث ما يسمح لك المغادرة من الخدمة الاحتياطية تقدم إلى إدارة التجنيد والتعبئة لاتخاذ اللازم.

٦ - اذا فقدت منك هذه الشهادة سارع بالاتصال بأجهزة السجلات لاستخراج بدلا منها.

٧ - لا تضع نفسك تحت طائلة القانون سواء بالتعاقد أو العمل بالجيوش الأجنبية بالصفة المدنية أو العسكرية دون الحصول على تصديق مسبق من الجهات المختصة.

التوقيع :
رتبة : لواء
اسم : أشرف مروان طاحون
مدير إدارة السجلات العسكرية

إدارة السجلات العسكرية - خدمة بمكاتب سجلات الغربية

نقابة الأطباء البيطريين
Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف
المهنة : طبيب بيطرى
رقم القيد : ٣١٦٧٩ / البحيرة
رقم الترخيص : ٣١٧٧٠
سارى حتى : ٢٠٢٠/٠١
الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام
د.حميدة الشيخ

اتحاد نقابات المهن الطبية
نقابة الأطباء البيطريين
www.egy-vet-synd.org

جامعة بنهـــا
مركز الشهادات الموحدة
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026

منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة بتـــاريخ : ١٩٨١/٩/٩
الجنســـية : مصرى الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكليـة : ٢٧/ ٤/ ٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥/ ٢٠٢١

الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥

المختص ماجد | رئيس القسم أ/أحمد البلاز | وكيل الكلية للدراسات العليا أ.د/ أمانى عبد الرحمن عباس | عميد الكلية

مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

(نموذج رقم ٦٠ ش ش)

جمهورية مصر العربية
وزارة الدفاع
إدارة شئون الضباط للقوات المسلحة
القيد : ٤٩٥٧
التاريخ: ٢٠٠٧/١٢/١٨ م

شهادة
خبرة لضابط إحتياط / مكلف

الى / م.أول احتياط غير مستدعى محمد محمود محمد يوسف

بناء على طلبكم المؤرخ فى ٢٠٠٧٠٣٠٠٤

بشأن إخطاركم بالأعمال التى مارستموها خلال فترة أستدعائكم بالقوات المسلحة

نفيدكم بأنكم قمتم بالأعمال الآتية:

الرقابة الصحية على الأغذية(اللحوم - الألبان ومنتجاتها) .

وذلك خلال الفترة من (٢٠٠٥/٠٩/٠١) الى (٢٠٠٦/١٢/٣١)

و القوات المسلحة تشكركم على خدمتكم و تتمنى لكم التوفيق .

لواء أ.ح / أحمد محمد أحمد نصر
مدير إدارة شئون الضباط للقوات المسلحة
بالتفويض التوقيع (.....)
عقيد / محمد ثروت حسان
رئيس فرع الشئون الشخصيه والخدمه الاجتماعيه

المضاعفات

المضاعفات نادرة ولكن الشديدة في هذا المرض هي قصور العمود الفقري.[١] يحدث هذا بسبب انسداد الشريان الفقري خلال مروره في الثقبة المستعرضة. تصبح مفاصل العمود الفقري صلبة في داء الفقار الرقبية. وهكذا تصبح الخلايا الغضروفية التي تحافظ على القرص محرومة من التغذية وتموت. قد تسبب النبتات العظمية الثانوية تضيقًا للأعصاب الشوكية، يظهر في شكل اعتلال الجذور.

الأسباب

ينتج داء الفقار عن سنوات من الضغط غير الطبيعي المستمر، الناتج عن خلع جزئي للمفصل، أو الإجهاد الناجم عن الرياضة، أو الصدمات الحادة أو المتكررة، أو الوضع السيئ، الذي يتم وضعه على الفقرات والديسك الموجودة بينها.

يتسبب الإجهاد غير الطبيعي في تكوين الجسم لعظام جديدة للتعويض عن توزيع الوزن الجديد. سيؤدي هذا الوزن غير الطبيعي الناتج عن إزاحة العظام إلى حدوث داء الفقار. يمكن أن تؤدي المواقف السيئة وفقدان الانحناءات الطبيعية للعمود الفقري إلى الإصابة بداء الفقار أيضًا. يمكن أن يصيب داء الفقار أي شخص في أي عمر؛ ومع ذلك، فإن كبار السن هم أكثر عرضة للإصابة.

التشخيص

تعد فحوصات التصوير بالرنين المغناطيسي والتصوير المقطعي المحوسب مفيدة في تشخيص الألم ولكنها ليست نهائية بشكل عام ويجب أخذها في الاعتبار مع الفحوصات البدنية والتاريخ.

العلاج

عادة ما يكون العلاج متحفظًا بطبيعته. تثقيف المريض حول تعديلات نمط الحياة، والعقاقير المضادة للالتهابات (NSAIDs) والعلاج الطبيعي، ورعاية تقويم العظام هي أشكال شائعة من الرعاية اليدوية التي تساعد في إدارة مثل هذه الحالات.[٥] قد تكون العلاجات البديلة الأخرى مثل التدليك وعلاج نقطة الاثر واليوجا والوخز بالإبر ذات فائدة محدودة. يتم إجراء الجراحة من حين لآخر

تتم الدعوة إلى الجراحة لعلاج اعتلال الجذور العنقي في المرضى الذين يعانون من آلام مستعصية أو أعراض تقدمية أو ضعف لا يتحسن مع العلاج المحافظ.

لا تزال المؤشرات الجراحية لداء الفقار العنقي (CSM) مع اعتلال النخاع مثيرة للجدل إلى حد ما ، ولكن «يوصي معظم الأطباء بالعلاج الجراحي بدلاً من العلاج المحافظ للاعتلال النخاعي المتوسط إلى الشديد»

قد يكون العلاج الطبيعي فعالاً لاستعادة نطاق الحركة والمرونة وتقوية القلب

ومع ذلك، فإن العلاج الطبيعي وعلاج العظام لا يمكن أن «يعالج» الانحطاط، ويرى بعض الناس أن الامتثال القوي لتعديل الوضعية ضروري لتحقيق أقصى فائدة من تخفيف الضغط والتعديلات وإعادة تأهيل المرونة.

ومع ذلك، فقد قيل أن سبب داء الفقار هو ببساطة الشيخوخة (على الرغم من أنه يمكن أن يكون موجودًا في أي عمر)، وغالبًا ما يمارس علاج تعديل الوضع من قبل أولئك الذين لديهم مصلحة مالية (مثل تعويض العمال)[٧] في إثبات أنه ناتج عن ظروف العمل والعادات الجسدية السيئة. فهم علم التشريح هو مفتاح الإدارة المحافظة لداء الفقار.

الجراحة

تهدف الإجراءات الجراحية الحالية المستخدمة في علاج داء الفقار إلى تخفيف علامات وأعراض المرض عن طريق تقليل الضغط في القناة الشوكية (جراحة تخفيف الضغط) و / أو عن طريق التحكم في حركة العمود الفقري (جراحة الدمج).

جراحة تخفيف الضغط: يمكن إجراء جراحة العمود الفقري من كلا المقاربة الأمامية والخلفية. يختلف النهج باختلاف الموقع وسبب ضغط الجذر. بشكل عام، تتم إزالة النباتات العظمية وأجزاء من القرص الفقري.

جراحة الدمج : تُجرى عندما يكون هناك دليل على عدم استقرار العمود الفقري أو سوء المحاذاة. يختلف استخدام الأجهزة (مثل البراغي السنية) في جراحات الاندماج عبر الدراسات.

مساعدباحث /محمدمحمودمحمديوسف وهيبه

اللهم إرزقنا الهجرة والزواج والذرية الموت
والدفن فى بقاع الحرمين الشريفين بسلطانك
ثم عبادك الصالحين
وبارك اللهم فى أعمالنا
وإكتب فيهم الإستفادة اللهم أمين

وسائل الإتصال /٠٠٢٠١٢١٠٩٧٣٠٧٤

E_MAIL : Godlimits@gmail.com

M0hammed_yousef@yahoo.com

Genome2000@yahoo.com

مرض الكبد الدهنى غير الكحولى

التشخيص :

لأن مرض الكبد الدهني غير الكحولي لا يُسبِّب أي أعراض في معظم الحالات، فإنه كثيرًا ما يتمُّ الانتباه إليه طبيًّا عندما تُشير اختبارات تُجرَى لأسباب أخرى إلى مشكلة في الكبد. ويُمكن أن يحدث ذلك إذا كانت حالة الكبد تبدو غير طبيعية عند الفحص بالموجات فوق الصوتية أو إذا كانت نتائج اختبار إنزيم الكبد لديكَ غير طبيعية.

وتشمل الاختبارات التي تُجرَى لمعرفة التشخيص وتحديد مدى شدة المرض ما يلي:

إختبارات الدم

الإجراءات التصويرية

فحص نسيج الكبد

إختبارات الدم:-

تعداد الدم الكامل
اختبارات إنزيمات الكبد ووظائف الكبد
اختبارات التهاب الكبد الفيروسي المزمن (التهاب الكبد A، التهاب الكبد C وغيرهما)
اختبار فحص الداء البطني
سكر الدم الصائم
الهيموغلوبين السكري A1C، والذي يوضح مدى ثبات سكر الدم لديك
فحص الدهون، والذي يقيس دهون الدم، مثل الكوليسترول والدهون الثلاثية

الإجراءات التصويرية:-

تشمل الإجراءات التصويرية المُستخدمة في تشخيص مرض الكبد الدهني غير الكحولي ما يلي:

- تصوير البطن بالموجات فوق الصوتية (الألتراساوند)،

- وهو الاختبار الأول الذي يُجرى عادةً في حالة الاشتباه في الإصابة بأمراض الكبد.

•فحص التصوير المقطعي المُحوسب أو التصوير بالرنين المغناطيسي للبطن.

• تفتقر هذه التقنيات القدرة على تمييز التهاب الكبد الدهني الكحولي من مرض الكبد الدهني غير الكحولي، ومع ذلك قد تظل تُستخدم.

•تصوير المرونة العابر، وهو نمط مُعزز من الموجات فوق الصوتية التي تقيس درجة تيبُّس الكبد لديك. يُشير تيبُّس الكبد لديك إلى وجود تليُّف أو تندُّب.

•تصوير المرونة بالرنين المغناطيسي، يعمل من خلال الدمج بين التصوير بالرنين المغناطيسي والموجات الصوتية لتكوين خريطة مرئية (مُخطط المرونة) تُظهر درجة تيبُّس أنسجة الجسم

فحص نسيج الكبد:-

إذا كانت الفحوصات الأخرى غير قاطعة، فقد يُوصي طبيبك بإجراء لأخذ عينة نسيج من الكبد (خزعة الكبد). تُفحص عينة النسيج في المختبر بحثًا عن علامات الالتهاب والتندُّب.

قد يكون إجراء خزعة الكبد غير مريح، وينطوي على خطورة منخفضة سيُراجعها الطبيب معك بالتفصيل. يُنفذ هذا الإجراء عن طريق إدخال إبرة عبر جدار البطن إلى الكبد.

العلاج:-

بداية العلاج عادةً هي فقدان الوزن من خلال الجمع بين اتِّباع نظام غذائي صحيٍّ، وممارسة التمارين الرياضية. يعالج فقدان الوزن الأسباب التي تُسهِم في الإصابة بمرض الكبد الدهني غير الكحولي. ومن الناحية المثالية، يكون فقدان ١٠% من وزن الجسم أمرًا مرغوبًا فيه، ولكن التحسن في عوامل الخطر يمكن أن يتضح إذا فقدْتَ من ٣ إلى ٥% من وزنك الأولي. وتمثل جراحة إنقاص الوزن أحد الخيارات أيضًا لأولئك الذين يحتاجون لفقد الكثير من الوزن.

قد تكون زراعة الكبد خيارًا لأولئك الذين لديهم تشمع بالكبد؛ نتيجة الإصابة بالتهاب الكبد الدهني غير الكحولي.

العلاجات المحتمَلة في المستقبل :-

لايوجد أي علاج بعقاقير معتمدة من إدارة الغذاء والدواء الأمريكية لمرض الكبد الدهني غير الكحولي، ولكن أُجريت دراسات على بعض الأدوية وكانت ذات نتائج واعدة.

بمساعدة طبيبكَ، يُمكنكَ اتخاذ خطوات لإحكام السيطرة على مرض الكبد الدهني غير الكحولي. يُمكنك:

إنقاص الوزن. في حالِ كنتَ مصابًا بزيادة الوزن أو السمنة، فقُمْ بتقليل عدد السعرات الحرارية التي تتناولها كل يوم، وقُمْ بزيادة نشاطكَ البدني من أجل إنقاص وزنك. تخفيض السعرات الحرارية هو مفتاح إنقاص الوزن والتحكُّم في هذا المرض. إن حاولتَ إنقاص الوزن في السابق، ولكنكَ لم تنجحْ، فاطلبِ المساعدة من طبيبك.

اخترْ نظامًا غذائيًّا صحيًّا. اتبعْ نظامًا غذائيًّا صحيًّا غنيًّا بالفواكه والخضراوات والحبوب الكاملة، وراقِبْ جميع السعرات الحرارية التي تتناولها.

مارِسِ الرياضة وكن أكثر نشاطًا. حاولْ تخصيص ٣٠ دقيقة على الأقل لممارسة التمرينات الرياضية معظم أيام الأسبوع.

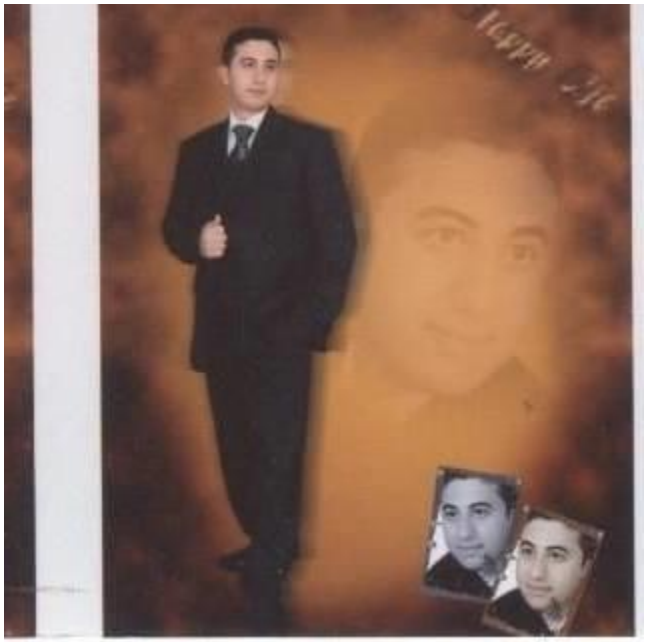
إذا كنتَ بصدد محاولة إنقاص وزنكَ، فستجد أن قيامكَ بالمزيد من التمارين أمر مفيد ونافع. ولكن إذا لم تمارسْ فعلًا التمارين بانتظام، فاحصُلْ على موافقة طبيبكَ أولًا وابدأْ ببطء.

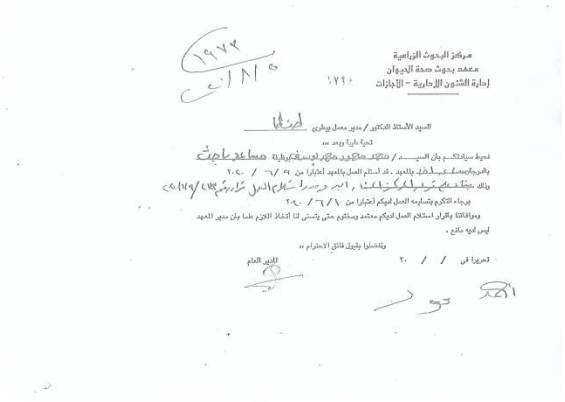
سيطِرْ على مرض السكري. اتَّبِعْ تعليمات طبيبكَ لتبقى مسيطرًا على مرض السكري الخاص بك. تناوَلِ الأدوية الموصوفة لكَ حسب الإرشادات وراقِبْ عن قرب سكر الدم لديك؟

قُمْ بتقليل الكوليسترول. إن اتباع نظام غذائي صحي يعتمد على النباتات وممارسة الرياضة، وتناول الأدوية يُمكن أن يُساعد في الحفاظ على الكوليسترول والدهون الثلاثية عند مستويات صحية.

حماية كبدك. تجنَّبِ الأشياء التي من شأنها أن تضع مزيدًا من الضغوط على الكبد. فعلى سبيل المثال، لا تتناوَل المشروبات الكحولية. اتَّبِعِ التعليمات الخاصة بالأدوية الموصوفةَ لكَ وأيضًا الأدوية المتاحة دون وصفة طبية. تشاوَرْ مع طبيبكَ قبل استخدام أي علاجات عشبية؛ لأنه ليس كل المنتجات العشبية آمنة.

فرط أوكسالات البول والداء الأوكسالى





جامعة بنهـــا
شهادة مؤقتة
تشهد كلية الطب البيطري
B 075026
منح السيد : محمد محمود محمد يوسف
المولود في : البحيرة
بتاريخ : ٩/٩/ ١٩٨١
الجنسية : مصرى
الرقم القومي: ٢٨١٠٩٠٩١٨٠٠٥١٦
درجة الماجستير في العلوم الطبية البيطرية تخصص:
"البكتريولوجيا والمناعة والفطريات"
وموضوع الرسالة: " تواجد ميكروبات التسمم الغذائى فى بعض أنواع الجبن الطرى فى السوق المحلى "
تاريخ اعتماد مجلس الكلية : ٢٧/ ٤ /٢٠٢١
تاريخ اعتماد مجلس الجامعة : ١٩/ ٥ /٢٠٢١
الكلية حاصلة على شهادة الاعتماد من الهيئة القومية لضمان جودة التعليم والاعتماد بالقرار رقم ١٩٩ بتاريخ ٢٠٢٠/١٠/٥
المختص
رئيس القسم
وكيل الكلية للدراسات العليا
عميد الكلية
ا/أحمد البار
أ.د/ أماني عبد الرحمن عباس
مستخرج من بيانات مركز الشهادات الموحدة بالجامعة بتاريخ ٢٠ - ٦ - ٢٠٢١

نظرة عامة

يحدث فرط أوكسالات البول عندما يحتوي البول على قدر كبير من الأوكسالات. وتُعد الأوكسالات مادة كيميائية طبيعية في الجسم، وتتوفر أيضًا في أنواع طعام معينة. ولكن قد يسبب وجود قدر كبير من الأوكسالات في البول مشاكل خطيرة.

وقد ينتج فرط أوكسالات البول عن اضطراب وراثي (جيني)، أو عن مرض معوي، أو تناول الكثير من الطعام الغني بمادة الأوكسالات.

وتعتمد صحة الكلى طويلة الأمد على التشخيص المبكر لفرط أوكسالات البول وعلاجها العاجل.

يحدث داء الأوكسالي بعد الإصابة بفشل الكلى للأشخاص الذين لديهم أسباب أولية، ومعوية للإصابة بفرط أوكسالات البول، وتراكم الأوكسالات المتزايدة في الدم. مما يؤدي إلى وجود رواسب للأُوكْسالات منها في الأوعية الدموية، وعظام الجسم وأعضائة.

الأعراض

غالبًا ما يكون أول أعراض فرط أوكسالات البول تَكوُّن حصوات الكلى. وقد تشمل أعراض حصوات الكلى ما يلي:

ألم حاد أو مفاجئ في الظهر

ألم مستمر لا يتوقف في المنطقة الواقعة أسفل الضلوع في الظهر (الخاصرة)

ظهور دم في البول

حاجة ملحة إلى التبول المتكرر

ألم مصاحب للتبول

القشعريرة أو حمى

الأسباب

تحدث حالة فرط أوكسالات البول عندما يكون هناك كمية كبيرة من مادة تسمى الأوكسالات في البول. يوجد عدة أنواع من حالة فرط أوكسالات البول:

فرط أوكسالات البول الأولي. فرط أوكسالات البول الأولي يعتبر حالة نادرة وراثية (جينية) تتواجد منذ الولادة. في هذا النوع، لا يُصنّع الكبد ما يكفي من بروتين معين (إنزيم) يمنع الإفراط في إنتاج الأوكسالات، أو أن الإنزيم لا يعمل بشكل صحيح.

يتم التخلص من الأوكسالات الزائدة من خلال كليتيك، في البول. الأوكسالات الإضافية يمكن أن تتحد مع الكالسيوم لتكوين حصوات وبلورات الكلى، والتي يمكن أن تتلف الكلى وتتسبب في توقفها عن العمل (الفشل الكلوي).

تتشكل حصى الكلى مبكرًا وغالبًا ما تسبب أعراضًا خلال الطفولة أو المراهقة. بسبب الكميات الكبيرة المنتجة من الأوكسالات، تفشل الكليتان بحلول مطلع فترة البلوغ وحتي أواسطها لدى العديد من الأشخاص المصابين بفرط أوكسالات البول الأولي. ولكن الفشل الكلوي يمكن أن يحدث في مرحلة مبكرة، مثل مرحلة الرضاعة، في حين أن الآخرين ممن يعانون فرط أوكسالات البول الأولي لا يتطور أبدًا الأمر لديهم إلى الفشل الكلوي. حتى الآن، حدد الخبراء ثلاثة أسباب وراثية مختلفة لفرط أوكسالات البول الأولي.

الداء الأوكسالي. يحدث الداء الأوكسالي إذا كنت مصابًا بفرط أوكسالات البول الأولي وكليتاك تتدهوران. ولأن جسمك لم يعد قادرًا على التخلص من الأوكسالات الإضافية، فإنها تبدأ في التراكم — أولاً في الدم، ثم في عينيك والعظام والجلد والعضلات والأوعية الدموية والقلب وغيرهم من الأعضاء. هذا يمكن أن يسبب مشاكل متعددة.

فرط أوكسالات البول المعوي. العديد من الأمراض المعوية، بما في ذلك مرض كرون ومتلازمة الأمعاء القصيرة الناجمة عن الإجراءات الجراحية، تزيد من امتصاص الأوكسالات من الأطعمة، والذي يمكن أن يزيد من كمية الأوكسالات التي تُفرز في البول.

فرط أوكسالات البول المتعلق بتناول الأطعمة عالية الأوكسالات. تناول كميات كبيرة من الأطعمة التي تحتوي على مستويات عالية من الأوكسالات يمكن أن يزيد من خطر إصابتك بفرط أوكسالات البول أو حصوات الكلى. اطلب من طبيبك أو أخصائي التغذية قائمة بالأطعمة عالية الأوكسالات. تجَنُب الأطعمة عالية الأوكسالات مهمًا للغاية إذا كنت مصابًا بفرط أوكسالات البول المعوي.

المضاعفات

شهادة تقديرات مواد السنوات الدراسية

الاسم: محمد محمود محمد يوسف تاريخ وجهة الميلاد: ١٩٨١/ م البحيرة

الدرجة الحاصل عليها الطالب: ((البكالوريوس فى العلوم الطبية البيطرية))

دور: مايو (٢٠٠٣) بتقدير عام: (جيد جدا) بنسبة (٧٥,٠٠%)

تاريخ موافقة مجلس الكلية: ٢٠٠٣/٧/١٥م مجلس جامعة طنطا: ٢٠٠٣/٧/٢٩م

جامعة

كفر الشيخ

شهادة تخرج

063342

تشهد كلية الطب البيطري

أن / محمد محمود محمد يوسف

المولود فى البحيرة بتاريخ ١٩٨١م

قد حصل على درجة البكالوريوس فى العلوم الطبية البيطرية

فى دور مايو عام ٢٠٠٣م بتقدير جيد جداً

مجموع تراكمى ٢٤٧٥/٣٣٠٠ النسبة المئوية ٧٥,٠٠%

وقد اعتمد مجلس الكلية نتيجة إمتحانه فى ٢٠٠٣/٧/١٥م

ومجلس الجامعة فى ٢٩ /٢٠٠٣/٧م بمقتضى قرار مجلس جامعة طنطا

الموظف المسئول مستاء المراجع مدير شئون الطلاب عميد الكلية

تحريراً فى ٢٠١٢/٥/٢٢م

نقابة الأطباء البيطريين

Egyptian Veterinarians Syndicate

الدكتور : محمد محمود محمد يوسف

المهنة : طبيب بيطرى

رقم القيد : ٣١٦٧٩ / البحيرة

رقم الترخيص : ٣١٧٧٠

ساري حتى : ٢٠٢٠/٠١

الرقم القومى : ٢٨١٠٩٠٩١٨٠٠٥١٦

الأمين العام

د.حميدة الشيخ

يمكن أن يؤدي إهمال علاج فرط أوكسالات البول الأولي في النهاية إلى تلف الكلى. وقد تتوقف الكلى عن العمل مع مرور الوقت. وفي بعض الأفراد، تكون هذه العلامة الأولى على الإصابة بالمرض.

وتتضمن علامات وأعراض فشل الكلى ما يلي:

انخفاض إخراج البول أو انعدامه كليًا

الشعور بالمرض أو التعب العام

فقدان الشهية والغثيان والقيء

شحوب لون البشرة المرتبط بفقر الدم

تورم اليدين والقدمين

يمكن أن يؤدي الداء الأوكسالي إلى مضاعفات متنوعة خارج الكلى، والتي تتضمن أمراض العظم وفقر الدم وقُرح الجلد ومشكلات القلب والعين، وفي الأطفال، فشل النمو الطبيعي.

التشخيص :-

ويقوم الطبيب بإجراء فحص طبي شامل والذي يتضمن مناقشة حول التاريخ الطبي والنظام الغذائي. تشمل فحوصات تشخيص فرط أوكسالات البول ما يلي

فحوصات البول، لقياس الأوكسالات ومستويات نواتج الأيض الأخرى في البول

فحوصات ألم، لبيان وظيفة الكلى ومستويات الأوكسالات في الدم

تحليل الحصوات، لتحديد مكونات حصوات الكلى التي تخرج مع البول أو بالإزالة الجراحية

الأشعة السينية على الكلى أو الموجات فوق الصوتية أو الفحص بالتصوير المقطعي المحوسب، بحثًا عن وجود أيّ حصوات في الكلى أو ترسبات أوكسالات الكالسيوم

وبعد الفحص الأول، قد ينصح الطبيب بالمزيد من الفحوصات لتأكيد التشخيص ومعرفة ما إذا كان المرض أصاب أجزاء أخرى من الجسم. قد تشمل تلك الاختبارات:

فحص الحمض النووي بحثًا عن الأسباب الوراثية (فرط أوكسالات البول الأولي)

خزعة الكلى بحثًا عن ترسبات الأوكسلات

مخطط صدى القلب لفحص ترسبات الأوكسلات في القلب

فحص العين لفحص ترسبات الأوكسالات في العين

خزعة نخاع العظم لفحص ترسبات الأوكسلات في العظام

خزعة الكبد لمعرفة حالات نقص الإنزيمات، حيث تلزم فقط في حالات نادرة عندما لا تكشف الفحوصات الوراثية عن سبب فرط أوكسالات البول

في حالة تشخيص إصابة المريض بفرط أوكسالات البول الأولي، يكون الأشقاء معرضين لخطر الإصابة بالمرض وينبغي خضوعهم للفحوصات. في حالة إصابة الأبناء بفرط أوكسالات البول الأولي، يمكن مراعاة إجراء الفحوصات الوراثية في حالة التخطيط لإنجاب مزيد من الأطفال. ويمكن للاستشاريين الوراثيين الطبيين المتمرسين أن يساعدوا في تقديم التوجيه بشأن القرارات والفحوصات.

العلاج:-

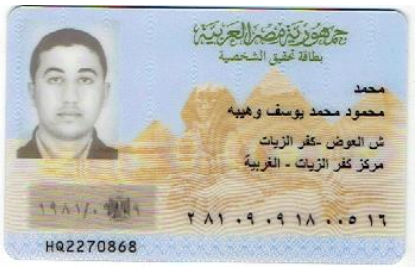
يعتمد العلاج على نوع مرض فرط أوكسالات البول وأعراضه وحدته ومدى جودة استجابتك للعلاج.

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

يمكن أن تكون جرعات فيتامين بى ستة فعالة في تقليل الأوكسالات في البول لدى بعض الأشخاص المصابين بفرط أوكسالات البول الأول.

تساعد مستحضرات السترات الفموية على منع تكوين بلورات أوكسالات الكالسيوم.

تناوُل كميات كبيرة من السوائل. إذا كانت الكليتان لا تزالان تعملان بصورة طبيعية، فمن المحتمل أن ينصحك طبيبك بشرب المزيد من الماء أو غيره من السوائل. إذ يعمل ذلك على غسيل الكلى ويمنع تراكم بلورات الأوكسالات ويساعد على منع تكوُّن حصوات الكلى.



التغيرات في النظام الغذائي. بوجه عام، تزداد أهمية العناية بنظامك الغذائي إذا كنت تعاني من فرط أوكسالات البول المعوي أو الغذائي. قد يوصي طبيبك بإدخال تغييرات إلى نظامك الغذائي، مثل تقليل الأطعمة التي تحتوي على نسبة عالية من الأوكسالات والحد من تناوُل الملح وتقليل البروتين الحيواني والسكر (شراب الذرة عالي الفركتوز). ويمكن أن تساعد مثل هذه التغييرات الغذائية على خفض مستويات الأوكسالات في البول. لكن قد لا تكون القيود الغذائية فعالة لدى جميع الأشخاص المصابين بفرط أوكسالات البول الأولي. اتبع توصيات طبيبك.

الأطعمة التي تحتوي على كميات كبيرة من الأكسالات

السبانخ.

اللوز.

الشمندر واللفت.

التوت.

زبدة الجوز.

المكسرات والكاجو.

الشوكولاتة والكاكاو.

ما أسباب تكون أوكسالات الكالسيوم؟

عدم الحصول على كمية كافية من السوائل. الإفراط في تناول نظام غذائي غني بالأوكسالات، أو البروتين، أو الملح. فرط نشاط جارات الغدة الدرقية. مرض التهاب الأمعاء.

السيطرة على حصوات الكلى

تنتشر حالات حصوات الكلى لدى المصابين بفرط أوكسالات البول ولكن لا تحتاج إلى العلاج دائمًا. إذا كانت الحصوات الكبيرة بالكلى تسبب الألم أو تعوق تدفق البول، فقد يحتاج المريض إلى إزالتها أو تفتيتها لتخرج مع البول.

الغسيل والزراعة

وفقًا لدرجة خطورة حالة فرط أوكسالات البول التي تعاني منها، ربما تصاب في نهاية المطاف بفقدان وظائف الكلى. ربما يكون غسيل الكلى مفيدًا بصورة مؤقتة، ولكنه لا يتوافق مع كمية الأوكسالات التي يتم إنتاجها. ربما تؤدي زراعة الكلى أو زراعة الكلى والكبد إلى التعافي من أنواع وراثية معينة من فرط أوكسالات البول (فرط أوكسالات البول من النوع الأول).

لتقليل كمية بلورات أوكسالات الكالسيوم المتكونة في كليتيك، قد يوصي طبيبك بواحد أو أكثر من هذه العلاجات:

BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE
DEPARTMENT OF BACTERIOLOGY, IMMUNOLOGY, AND MYCOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Youssef Wahhabi

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

Pure microbiology

Branches of microbiology

Bacteriology: the study of bacteria.
Mycology: the study of fungi.
Protozoology: the study of protozoa.
Phycology/algology: the study of algae.
Parasitology: the study of parasites.
Immunology: the study of the immune system.
Virology: the study of viruses.
Nematology: the study of nematodes.

https://en.wikipedia.org/wiki/Branches_of_microbiology

GENERAL

BACTERIOLOGY, IMMUNOLOGY, MYCOLOGY

INTRODUCTION TO MICROBIOLOGY

MICROBIOLOGY

IS DEFINED AS

(THE STUDY OF ORGANISMS TOO SMALL TO BE CLEARLY SEEN BY THE NAKED EYES).

THESE ORGANISMS INCLUDE VIRUSES, BACTERIA, ALGEA, FUNGI, ARCHAEA AND PROTOZOA. WHEN STUDYING MICROORGANISMS , MICROBIOLOGISTS ARE CONCERNED WITH CHARACTERISTICS AND FUNCTIONS SUCH AS MORPHOLOGY ,CYTOLOGY, PHYSIOLOGY , ECOLOGY , TAXONOMY , GENETICS , AND MOLECULAR BIOLOGY .

TAXONOMY التصنيف الحيوى ECOLOGY علم البيئة

Leeuwenhoek
(Live 1632 -1723)
is the first person to use the microscopes to observe microbes: he made small handheld microscopes: he called ((microorganisms as animalcules)).

خلايا بدائيات النوى خلية بدائية النواة Procaryotes

TAXONOMY AND CLASSIFICATION OF PROCARYOTES

HEAKEL (1866) WAS THE FIREST TO CREATE ANATURAL KINGDOM FOR MICROORGANISMS , HE PLACED ALL UNICELLULAR (MICROSCOPIC) IN ANEW KINGDOM CALLED PROTISTA , SEPERATED FROM PLANTS (PLANTAE) AND ANIMALS (ANIMALIA) , WHICH WERE MULTICELLULAR (MACROSCOPIC) ORGANISMS .

PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS
PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS
ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

IN (1950) : THE DEVELOPMENT OF ELECTRON MICROSCOPE

THE ELECTRON MICROSCOPE MORE ONE MILLION POWERFUL THAN THE LIGHT MICROSCOPE.

SO BY THE ELECTRON MICROSCOPE THEY FIND ANOTHER KINGDOM CALLED MONERA (MONERES) : INCLUDES MICROSCOPIC UNICELLULAR ORGANISMS BUT NOT HAVE THE NUCLEAR MEMBRANE .

PROTISTA :
REMAINED AS AKINGDOM OF UNICELLULAR EUCARYOTIC MICROORGANISMS .

WHITTAKER : REFINED THE ECOSYSTEM INTO FIVE KINGDOMS IN (1967) BY IDENTIFYING THE FUNGI AS ASEPERATE MULTICELLULAR EUOCARYOTIC KINGDOM OF ORGANISMS , DISTINGUISHED BY THEIR ABSORPTIVE MODE OF HETEROTROPHIC MODE OF NUTRITION .

يتكون النظام البيئى إلى خمس مملكات حيوية :

THE FIVE KINGDOMS OF THE ECOSYSTEM :

1-PROTISTA : UNICELLAR MICROSCOPIC ORGANISMS

2-PLANTAE : MULTICELLULAR MACROSCOPIC ORGANISMS

3-ANIMALIA : MULTICELLULAR MACROSCOPIC ORGANISMS

4- MONERA : UNICELLULAR MICRORGANISMS NOT HAVE NUCLEAR MEMBRANE.

5- FUNGI : MULTICELLULAR OF EUCRAYOTIC ORGANISMS ITS LENGHTH 4 MICROMETERS IN LENGTH .

BACTERIA IS 1 MICROMETERS IN LENGTH

FUNGI IS 4 MICROMETERS IN LENGTH

FIVE KINGDOMS OF THE ECOSYSTEM AS FELLOW :

ANIMALIA :INCLUDES VERTEBRATES AND ARTHROPODES

PLANTEA : INCLUDES GREEN ALGEA

FUNGI : INCLUDES MOLDS AND YEASTS

MONERA : INCLUDES ARCHAEA ,BACTERIA,MYCOPLASMA

PROTESTA : INCLUDES RED ALGEA , AMOEBAS, WATER MOLDS .

What are 5 characteristics of fungi?

Characteristics of Fungi

Fungi are eukaryotic, non-vascular, non-motile and heterotrophic organisms.

They may be unicellular or filamentous.

They reproduce by means of spores.

They Have Two Types Of Reproduction Sexually And Asexually Reproduction .

Fungi exhibit the phenomenon of alternation of generation.

Fungi lack chlorophyll and hence cannot perform photosynthesis.

BACTERIAL MORPHOLOGY

PLEASE CHANGES THE PICTURES TO BE ELECTRONIC MICROSOPIC RECOGNITIONS

PROKARYOTIC CELLS ARE GENERALLY MUCH SMALLER THAN EUKARYOTIC CELLS

BACTERIA REMARKKABLY VARY IN SIZES , SHAPES AND ARRANGEMENT

MOST OF BACTERIA COME IN BASIC FOUR SHAPES :

1-COCCI 2- ROD OR BACILLI 3-ARCH

4-SPIRAL

1-COCCUS

THE COCCI ARE SPHERICAL OR OVAL BACTERIA HAVING ONE OF SEVERAL DISTICNT ARRANGEMENTS :

1-DIPLOCOCCUS :PAIR OF COCCI

2-STREPTOCOCCUS : CHAIN OF SEPERATE COCCI

3-TETRAD : SQUARE OF FOUR COOCI

4-SARCINA :CUBE OF 8 COCCI

5-STAPHYLOCOCCUS:COCCI IN IRREGULAR , OFTEN GRAP LIKE CLUSTE

2-BACILLUS :

BACILLI ARE ROD SHAPED BACTERIA , THEY DEVIDED IN ONE PLANE PRODUCING ONE OF THE FOLLOWING :

1-BACILLIUS : ASINGLE BACILLUS (CYLINDRICAL)

2- COCCO BACILLUS HAVE ABULGING MOSTLY IN THE MIDDLE

3-STREPTO BACILLI : ACHAIN OF SEPERATED BACILLUS TO BE FACILITATING CONGUGATION

4-SPIRAL : AHELICAL OR CORKSCREW –SHAPED BACTERIUM .SPIRALS COME IN 1 OF 3 FORMS :

لولبى

1-VIBRIO : CURVED OR COMA SHAPED ROD

2-SPIRILLUM : THICH RIGID SPIRAL

3-SPIROCHETE : THIN FLEXIBLE SPIRAL

PROCARYOTIC CELL STRUCTURES AND FUNCTIONS

THE WORD PROCARYOTE WILL BE USED IN AGENERAL SENSE TO INCLUDE BOTH BACTERIAE AND ARCHAEA
(MYCOPLASMA IN ASEPERATE TAXONOMY)
ARCHAE ARE MICROORGANISMS THEY ARE NO MORE RELATED TO BACTERIA THAN TO EUKARYOTES . THEY SHARE SOME TRAITS WITH BACTERIA ,SUCH AS HAVING ASINGLE CIRCULAR MOLECULE OF DNA ,THE PRESCENSE OF MORE MOBILE PIECES OF GENATIC MATERIALES CALLED PLASMIDS ,SIMILAR ENZYMES FOR PRODUCING COPIES OF DNA . HOWEVER THEIR METHODS OF PROTIEN PRODUCTION AND ORGANIZATION OF THEIR GENATIC MATERIAL BEARS MORE SIMILARITY TO EUBACTERIAL CELLS .
ARCHAE INCLUDES MORE ARELATIVELY SMALL NUMBERS OF MICROORGANISMS THEY ARE INHABITING ENVIRONMENTS WHICH ARE TOO HARSH FOR OTHER MICROBES.SUCH AS ENVIRONMENTS INCLUDES HOT ,MOLTEN VENTS AS AT THE BOTTOM OF OCEANS , AND THE HIGHLY SALT WATER OF THE GREAT SALT LAKE AND THE DEAD SEA.

البحر الميت – البحيرة المالحة الأمريكية العظيمة ولاية أوتاها كلاهما مالحان

فتحات مصهورة --- لقدذكرت من علامات الساعة

الكبرى جفاف بحيرة طبريا وهى من أعذب وأحلى مياه العالمين

البحر المميت الأردن قوم لوط العرب

البحيرة المالحة الأمريكية بولاية أوتاها ليس لها مسمى فاحش

UUWD89J308ULRP4

الشكوى الإدارية فى ملفات الرقابة الإدارية المصرية

DIFFERENCES BETWEEN PROKARYOTE AND EUKARYOTE AND ARCHAEA :

THE COMPARISONS WILL COVER THIS POWER POINTS :

1-CELL CONFIGURATIONS 2-NUCLAR MEMBRANE 3-NUMBER OF CHROMOSOME 4-CHROMOSOME TOPOLOGY 5-MURIEN IN CELL WALL 6-CELL MEMBRANE LIPIDS 7- CELL MEMBRANE STEROLS 8- ORGANELLES ESPECIALLY MITOCHONDRIA AND CHLOROPLASTS) 9-RIBOSOMES SIZES 10- CYTOPLASMIC STREAMING 11-MEIOSIS AND MITOSIS) 12- TRANSCRIPTIONS AND TRANSLATIONS COUPLED 13- AMINO ACID INITIATING PROTEIN SYNTHESIS

14- PROTEIN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHENICOL

15- PROTEIN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN

DIFFERENCES BETWEEN EUKARYOTE, PROKARYOTE,ARCHAEA			
PROPERITY	BIOLOGICAL DOMAIN		
	EUCARYA	BACTERIA	ARCHAEA
CELL CONFIGURATION	EUCARYOTIC	PROCARYOTIC	PROCARYOTIC
NUCLEAR MEMBRANE	PRESENT	ABSENT	ABSENT
NUMBER OF CHROMOSOMES	MORE THAN ONE	ONE	ONE
CHROMOSOME TOPOLOGY	LINEAR	CIRCULAR	CIRCULAR
MUREIN IN CELL WALL	-	+	-
CELL MEMBRANE LIPIDS	ESTER-LINKED GLYCERIDES UNBRANCHED POLYUNSATURATED	ESTER-LINKED GLYCERIDES: UNBRANCHED SATURATED OR MONOUNSATURATED	ETHER –LINKED BRANCHED: SATURATED
CELL MEMBRANE STEROLS	PRESENT	ABSENT	ABSENT

ORGANELLES (MITOCHONDERIA AND CHLOROPLASTS)	PRESENT	ABSENT	ABSENT
RIBOSOME SIZE	80S	70S	70S
CYTOPLASMIC STREAMING	+	–	–
MEIOSIS AND MITOSIS	PRESENT	ABSENT	ABSENT
TRANSCRIPITION AND TRANSLATION COUPLED	–	+	+
AMINOACID INITIATING PROTIEN SYNTHESIS	METHIONINE	N-FORMYLE METHIONINE	METHIONINE
PROTIEN SYNTHESIS INHIBITING BY STREPTOMYCIN AND CHLORAMPHINCOL	–	+	–
PROTIEN SYNTHESIS INHIBITED BY DIPHTHERIA TOXIN	+	–	+

BACTERIAE

LECTURES WRITTEN AND PRESENTED BY

DOCTOR

MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA

BACTERIAL STRUCTURES

INCLUDES :

1- ESSENTIAL BACTERIAL STRUCTURES

2- NON ESSENTIAL BACTERIAL STRUCTURS

1- ESSENTIAL BACTERIAL STRUCTURES

INCLUDES :

1-CELL WALL AND

2-PROTOPLASM

2-PROTOPLASM INCLUDES :

A-CYTOPLASM AND

B-CELL MAMBRANE

A-CYTOPLASM INCLUDES :

CHROMOSOMES ,MESOSOMES,RIBOSOMES, INCLUSIONS BODIES.

2-NON ESSENTIAL BACTERIAL STRUCTURES :

INCLUDES :

CAPSULES,FLAGELLAE,PILI,
SPORES,
PLASMIDES.

1-ESSENTIAL BACTERIAL STRUCTURES :

1-CELL WALL :

1-ONE OF THE MOST IMPORTANT PARTS OF THE PROKAYOTIC CELL.

2-MOST OF PROCAYOTES HAVE ARIGID CELL WALL.

3-THE CELL WALL IS CELL MADE OF PEPTIDOGLYCANS: POLYMER OF PEPTIDES AND GLYCANS.

PEPTIDES (TYPICAL 4 AMINOACIDES LONG ,CROSS-LINKED TO OTHER CHAINS)

GLYCANS (MADE OF ALTERNATING AMINO SUGARS)

4- PEPTIDOGLYCAN IS AHUGE POLYMER OF INTERLOCKING CHAINS OF IDENTICAL MONOMERS.

5- THE BACKBONE OF PEPTIDOGLYCAN MOLECULES COMPOSED OF TWO DERIVATIVES OF GLUCOSE :
N-ACETYLE GLUCOSAMINE(NAG)
AND N-ACETYLMURAMIC ACID (NAM)
THE (NAG) AND (NAM) STRANDS ARE CONNECTED BY INTERPEPTIDE BRIDGES

6-FUNCTIONS OF CELL WALL :

1-SUPPORTS THE WEAK CYTOPLASMIC MEMBRANE AGAINST INTERNAL OSMOTIC PRESSURE .

2-RESPONSIBLE FOR THE SHAPE OF BACTERIAE AND THEIR GRAM STAIN REACTIONS .

3-INTEGRITY OF THE CELL WALL ESSENTIAL FOR VIABILITY OF BACTERIAE .

4-PLAY ROLE IN CELLS DIVISIONS AND ANTIGENICITY OF THE BACTERIAE .

IN THE GRAM POSITIVE BACTERIAE THOSE RETAIN CRYSTAL VIOLET DYE WHEN SUBJECTED TO THE GRAM STAINING PROCEDURES , THE CELL WALL CONSISTS OF SEVERAL LAYERS OF PEPTIDO GLYCAN . TEICHOIC ACIDS RUNING PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.
TEICHOIC ACIDS IS AGROUP OF MOLECULES WHICH ARE UNIQUE TO GRAM POSITIVE CELL WALL .

STRUCTURE OF THE GRAM POSITIVE BACTERIAL CELL WALL .THE CELL WALL IS RELATIVELY THICK AND CONSISTS OF MANY LAYERS OF PEPTIDO GLYCANS INTERSPERSED WITH TECHOIC ACIDS THAT RUN PERPENDICULAR TO THE PEPTIDOGLCAN SHEETS.

IN THE GRAM NEGATIVE BACTERIAE (WHICH DO NOT RETAIN CRYSTAL VIOLET DYE EXCEPT ALITTLE) :

THE CELL WALL COMPOSED OF ASINGLE LAYER OF PEPTIDO GLYCANS SURROUNDED BY AMEMBRANOUS STRUCTURE CALLED THE OUTERMEMBRANE.

THE OUTER MEMBRANE OF GRAM NEGATIVE BACTERIAE INVARIABLY CONTAINS AUNIQUE COMPONENT,LIPOPOLYSACCHARIDES (LPS OR ENDOTOXIN), WHICH IS TOXIC IN ANIMALS.

STRUCTURE OF THE GRAM NEGATIVE- BACTERIAE CELL WALL.

THE WALL IS RELATIVELY THIN AND CONTAINS MUCH LESS PEPTIDO GLYCANS THAN THE GRAM POSITIVE BACTERIAL WALL. ALSO,TEICHOIC ACIDS ARE ABSENT. HOWEVER, THE GRAM NEGATIVE-CELL WALL CONSISTS OF AN OUTERMEMBRANE THAT IS OUTSIDES OF THE PEPTIDO GLYCAN LAYER.THE OUTERMEMBRANE IS ATTACHED TO THE PEPTIDO GLYCAN SHEET BY AUNIQUE GROUP OF LIPOPROTIEN MOLECULES.

THE PROFILES OF THE CELL ENVELOPE OF THE GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE.

THE GRAM POSITIVE WALL IS AUNIFORMLY THICK LAYER EXTERNAL TO THE PLASMA MEMBRANE . IT IS COMPOSED MAINLY OF PEPTIDOGLYCAN (MEURIN).

THE GRAM NEGATIVE WALL

APPEARS THIN AND MULTILAYERED.

IT IS CONSIST OF ARELATIVELY THIN PEPTIDOGLYCAN SHEET BETWEEN THE PLASMA MEMBRANE AND APHOSPHOLIPID-LIPOPOLYSACCHARIDE OUTERMEMBRANE.

THE SPACE BETWEEN THE INNER PLASMA MEMBRANE AND OUTER MEMBRANE WHERE IN THE PEPTIDOGLCAN RESIDES IS CALLED PERIPLASM.

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

2-THE CELL MEMBRANE (PLASMA MEMBRANE):

1-BACTERIAL MEMBRANES COMPOSED OF 40% TO 60% PROTIEN.BACTERIAL PLASMA MEMEBRANES USUALY HAVE AHIGHER PROPORTION OF PROTIEN THAN DO EUCARYOTIC MEMBRANES,PERSUMABLY BECOUSE THEY FULFIL SO MANY DIFFERENT FUNCTIONS THAT ARE CARRIED OUT BY OTHER ORGANELLE MEMBRANES IN EUCARYOTES.

FUNCTIONS OF-Be- CELL MEMBRANE:

1-OSMOTIC BARRIER FOR MAINTAINING DIFFERENCE IN SOLUTE CONTENT BETWEEN CYTOPLASM AND EXTERNAL ENVIRONMENT.

2-PERMIT PASSIVE DIFFUSION INWARD FROM OUTWARD OF WATER AND LIPID SOULABLE MATERIAL.

3-SELECTIVE TRANSPORT OF SPECIFIC NUTRIENT SOLUTE IN TO THE CELL.

4-ENZYMATIC ACITIVITY AS IT CONTAINS PERMEASE,RESPIRATORY,CYTOCHROME AND POLYMERZING ENZYMES.

5-ENERGY GENERATING FUNCTIONS,INVOLVING RESPIRATORY AND PHOTOSYNTHETIC ELECTRON TRANSPORT SYSTEMS,ESTABLISHMENT OF PROTON MOTIVE FORCE, AND TRANSMEMBRANOUS, ATP-SYNTHESIZING ATPase.

6-COORDINATIONS OF DNA REPLICATIONS AND SEGREGATION WITH SEPTUM FORMATION AND CELL DIVISION.

2-THE PERIPLASM :

IS ASPACE BETWEEN PLASMA MEMBRANE AND CELL WALL.

IT IS VISIBLE IN GRAM NEGATIVE AND DIFFICULT IN GRAM POSITIVE BACTERIAE.

IT CONTAIN VARIOUS HYDROLYTIC ENZYMES AND BINDING PROTIENS THAT SPECIFICALLY BIND SUGARS ,AMINOACIDS AND INORGANIC IONS ,THESE IONS AND PROTIENS THAT AID IN TRANSPORT VARIOUS COMPOUNDS INTO AND OUT BACTERIAL CYTOPLASM.

3-THE CYTOPLASM :

1-THE CYTOPLASM OF BACTERIAL CELL IS VISCOUS WATERY SOLUTION CONTAIN ORGANIC AND INORGANIC SUBSTANCES AND RIBOSOMES.
2-IT HAS NO MITOCHONDERIA,NO ENDOPLASMIC RETICULUM, NO GOLGI APPARATUS.
3-IT COMPOSED FROM RNA.
4-IT CONTAINS BACTERIAL CHROMOSOMES (NECLEOID),RIBOSOMES,INCLUSION GRANULES AND MESOSOMES.

4-THE BACTERIAL CHROMOSOMES (NECLOID):

IT COMPRISES ACLOSED LOOP OF DOUBLE STRANDED SUPERCOILED DNA.(NO NUCLEAR MEMBRANE).IN ADDITION ,SOME BACTERIAE CONTAIN ADDITIONAL DNA IN THE FORM OF SMALL ,SELF REPLICATING EXTERA-CHROMOSOMAL ELEMINTS CALLED PLASMIDS.THESE DO NOT CARRY ANY GENES ESSENTIAL FOR GROWTH AND REPRODUCTIONS,AND THUS THE CELL MAY SERVIVE WITHOUT THEM. THE PLASMIDS CAN BE VERY IMPORTANT HOWEVER,AS THEY MAY INCLUDE GENES ENCODING TOXINS OR RESISTANCES TO ANTIBIOTICS ,AND CAN BE PASSED FROM CELL TO CELL.

5-RIBOSOMES:

THESE ARE THE SITES OF PROTIENS SYNTHESIS.ALTHOUGH THEY CARRY OUT ASIMILAR FUNCTIONS,THE RIBOSOMES OF PROCARYOTIC CELLS ARE SMALLER AND LIGHTER THAN THIER EUCARYOTIC CELL.
PROCARYOTIC RIBOSOMES ARE 70 S ,WHILE THOSE OF EUCARYOTES ARE 80 S.

6-MESOSOMES:

IT IS ACONVOLUTED BODY INVAGINATED FROM CYTOPLASMIC MEMBRANE INTO CYTOPLASM.
1-ENTER IN THE MECHANISM OF CELL DIVISION.
2-HAS AROLE IN SPORULATION OF BACTERIA.
3- ACT AS AMITOCHONDERIA OF EUCARYOTIC CELL.
4-HAS AROLE IN RESPIRATION OF BACTERIA.
5-EXCRETE MATERIALS FROM CYTOPLASM TO EXTERIOR.

7-INCLUSIONS BODIES :

1-IT WERE ASTORED RESERVES OF NUTRIENTS OF AGGREGATES OF EXCESS SUBSTANCES .

2-IT VARRIES ACCORDINDG TO TYPE OF BACTERIAL STRUCTURE OR GENOMES.

E.G: FOR EXAMPLES:

a-VOLUTIN (METACHROMIC GRANULES) IN PASTEURELLA SPP.

b-LIPIDS GRANULES IN TUBERCLE BACILLI.

NON ESSENTIAL STRUCTURES

1-CAPSULES AND -S LAYERS

2-FELAGELLAE

3-PILI

4-SPORE

5-PLASIMDS .

1-CAPSULES AND S LAYERS :

A-SOME BACTERIA HAVE ALAYER OF MATERIAL LYING OUTSIDE THE CELL WALL

B-WHEN THE LAYER IS WELL RECOGNIZED AND EASILY WASHED OFF IT IS CALLED ATRUE CAPSULE.

C-ATRUE CAPSULE IS A DISCRETE DETECTABLE LAYER OF POLYSACCHARIDES DEPOSITED OUTSIDE THE CELL WALL.

D-ALESS DISCRETE STRUCTURE OR MATRIX WHICH EMBEDS THE CELL CALLED ASLIME LAYER OR BIOFILM.

E-CAPSULES AND SLIME LAYERS USUALLY ARE COMPOSED OF POLYSACCHARIDES .

F-BUT THEY MAY BE CONSTRUCTED FROM OTHER MATERIALS .

G-FOR EXAMPLE BACILLUS ANTHRAX HAVE ACAPSULE OF POLY-GLUTAMIC ACIDS .

H-CAPSULES ARE CLEARLY VISIBLE BY IN LIGHT MICROSCOPE.

I-MANY GRAM POSITIVE BACTERIAE AND GRAM NEGATIVE BACTERIAE HAVE AREGULARLY STRUCTURED LAYER AN S LAYER ON THIER SURFACE . THE S-LAYER HAS APATTERN SOMETHING LIKE FLOOR TILES AND IS COMPOSED OF PROTIEN OR GLYCOPROTIEN.

N.B THERE AN ACONFESSION THAT THERE AN ELECTRON MICROSOPE IMPLENTATIONS IN THE UNIVERSITY BOOK.

J-BACTERIAL CAPSULES OUTLINED BY INDIA INK VIEWED BY LIGHT MICROSCOPY .THIS ATRUE CAPSULE ,ADISCRETE LAYER OF POLYSACCHARIDE SURROUNDING THE CELLS.SOMETIMES BACTERIAL CELLS ARE EMBEDDED MORE RANDOMLY IN APOLYSACCHARIDES MATRIX IN ASLIME LAYER OR BIOFILM.

FUNCTIONS OF CAPSULES :

A-PROTECT THE CELL WALL AGAINST ANTIBACTERIAL AGENTS .
B-PROTECT THE BACTERIAL CELLS AGAINST INGESTION BY THE PHAGOCYTIC CELLS .
C-RESPONSIBLE OF VIRULENCE OF THE ORGANISMS
D-THE S -LAYER CAN FUNCTION AS AN ADHESION ,ENABLING THE BACTERIUM TO ADHERE TO HOST CELL MEMEBRANES AND ENVIRONMENTAL SURFACES IN ORDER TO COLONIZES .
E-MANY OF THE CELL –ASSOCIATED PROTIEN ADHESINS USED BY PATHOGENS ARE COMPONENTS OF THE S-LAYER.

2-FLAGELLA :

A-MOST MOTILE BACTERIA MOVE BY USE OF FELAGELLA (S.FLALGELLUM) ,
B-THREADS LIKE LOCOMOTOR APPENDAGES EXTENDING OUTWARD FROM THE PLASMAMEMBRANE.
C-THEY ARE SLENDER , RIGID STRUCTURES ,ABOUT 20 NM ACROSS AND UP TO 15 OR 20 MICRO METER LONG .
D-FLAGELLA ARE SO THIN THEY CAN NOT BE OBSERVED DIRECTLY WITH –A BRIGHT FIELD MICROSCOPE.
E-BUT MUST BE STAINED WITH ESPECIAL TECHNIQUES DESIGNED TO INCREASE THEIR THICKNESS.

F-BACTERIAL SPECIES OFTEN DIFFER DISTINCTIVELY IN THEIR PATTERNS OF FLAGELLAE DISTRIBUTIONS :

1-MONOTRICHOUS BACTERIA (TRICHOUS MEANS HAIR) HAVE ONE FELAGELLUM:IF IT IS LOCATED AT THE END IT IS SAID TO BE POLAR FLAGELLUM

2-AMPHITRICHOUS BACTERIA (AMPHI MEANS ON BOTH SIDES) LE FELAGELLUM ز HAVE ASINGLE FELAGELLUM ON THE BOTH POLE. ON THE BOTH POLE>BOTH POLE BE POLAR FLAGELLUM E PLASMAMEMBRANE MEBRANE LE ,ADISCRETE LAYER OF POLYSACCHARIDE SURR

3-LOPHOTRICHOUS BACTERIA (LOPHO MEANS TUFT) HAVE ACLUSTER OF FELAGELLA AT ONE OR BOTH ENDS .

4-PERITRICHOUS (PERI MEANS AROUND) BACTERIA HAVE FLAGELLA THAT ARE SPREAD FAIRLY EVENLY OVER THE WHOLE SURFACE . FELLAGELATION PATTERNS ARE VERY USEFUL IN IDENTIFYING BACTERIA.

G-FELAGELLAR ULTRASTRUCTURE:

A-THE LONGEST AND MOST OBIVIOUS PORTION IS THE FILAMENT ,WHICH EXTENDEDS FROM THE CELL SURFACE TO THE TIP.

b-abasal body is embedded in the cell :the body has four rings connected to a centeral rod . the outer l and p rings associated with the lipopolysaccharides and peptidoglycan layers ,respectively .

d-ashort curved segment ,the hook ,links the filament to to its basal bodies and acts as aflexible coupling.

THE FILAMENT IS AHOLLOW ,RIGID CYLINDER CONSTERUCTED OF ASINGLE PROTIEN CALLED FLAGELLIN .

3-FIMBRIAE AND PILI :

FIMBRIAE AND PILLI ARE SHORT-HAIR LIKE STRUCTURES ON THE SURFACES OF PROCARYOTIC CELLS .LIKES FELAGELLA ,THEY ARE COMPOSED OF PROTIEN .FIMBRIAE ARE SHORTER AND STIFFER THAN FELAGELLA AND SLIGHTELY SMALLER IN DIAMETER .GENERALLY ,FIMBERIAE HAVE NOTHING TO DO WITH BACTERIAL MOVEMENT (THERE ARE EXCEPTIONS, E.G . TWITCHING, MOVEMENT ON PSEUDOMONAS).

N.B FIMBERIAE ARE MOST COMMON IN GRAM NEGATIVE BACTERIAE , BUT OCCUR IN SOME GRAM POSITIVE BACTERIAE AS WELL.

FIMBRIAE ARE MOST OFTEN INVOLVED IN ADHERENCE OF BACTERIA TO SURFACES ,SUBSTRATES AND OTHER CELLS OR ISSUES IN NATURE. IN E.CLOI , ASPECIALIZED TYPE OF PILUS , THE F OR SEX PILUS, APPARENTELY STABLIZES MATING BACTERIA DURING THE PROCESS OF CONJUGATION , BUT THE FUNTION OF THE SMALLER , MORE NUMEROUS COMMON PILLI ARE QUITE DIFFERENT .

COMMON PILI (ALMOST ALWAYS CALLED FIMBRIAE) ARE USUALLY INVOLVED IN SPECIFIC AHERENCE (ATTATCHMENT) OF PROCARYOTES TO SURFACES IN NATURE.

IN THE MEDICAL SITUATIONS , THEY ARE MAJOR DETERMINANTS OF BACTERIAL VIRULENCE BECOUSE THEY ALLOW PATHOGENS TO ATTACH TO (COLONIZE) TISSUES AND OR TO RESIST ATTACK BY PHAGOCYTIC WHITE BLOOD CELLS . FOR EXAMPLE , PATHOGENIC NEISSERIA GONORHOEAE ADHERES SPECIFICALLY TO THE HUMAN CERVICAL OR URETHERAL EPITHELIUM BY MEANS OF FIMBERIAE : ENTEROTOXIGENIC STRAINS OF ESCHERICHIA COLI ADHERE TO THE MUCOSAL EPITHELIUM OF THE INTESTINE BY MEANS OF SPECIFIC FIMBRIAE .

PROPERTY	FLAGELLA	PILI
COMPOSITION	FLAGELLIN, APROTIEN	PILIN, APROTIEN
OCCUERENCE	MANY BACTERIAL RODS,BUT FEW COCCI	MANY GRAM NEGATICE AND COCCI
FUNCTION	MOTION	CONJUGATION,ATTACHMENT
SHAPE	LONG AND THIN	SHORT AND STIFF
ORIGIN	HOOK LIKE INSERTION TO BASAL GRANULE INSIDE CELL WALL	CYTOPLASM

4- THE BACTERIAL END SPORES:

1-ANUMBER OF GRAM POSITIVE BACTERIA CAN FORM ASPECIAL RESISTANT , DORMANT STRUCTURE CALLED AN END SPORES .
2-END SPORES DEVELOP WITHIN VEGETATIVE BACTERIAL CELLS OF SEVERAL GENERA
3-SEVERAL GENERA INCLUDES : 1- BACILLUS LIKES ANTHRAX AND CLOSTRIDIUM (RODS) AND 2- COCCI LIKES SPOROSARCINA
3- AND OTHERS .

4- THESE STRUCTURES ARE EXTRA ORDINARILY RESISTANT TO INVIRONMENTAL STRESSES SUCH AS HEAT,ULTRA VIOLET, RADIATION,GAMMA RADIATIONS, CHEMICAL DISINFECTANTS,DESICCATIONS,

5- IN FACTS ,SOME ENDO SPORES HAVE REMAINED VIABLES FOR AROUNDING 100,000 YEARS.

6-BECOUSES OF THEIR RESISTANSE AND INFACT THAT SEVERAL SPECIES OF ENDOSPORES –FORMING BACTERIA ARE DANGEROUS PATHOGENS , ENDOSOPRES ARE OF GREAT PRACTICAL IMPORTANCE IN FOOD,INDUSTERIAL ,AND MEDICAL MICROBILOGY ,

7-THIS BECOUSE ENDOSPORES OFTEN RESIST SURVIVE BOILING FOR AN HOUR OR MORE.

8-SPORES POSITIONS IN THE MOTHER CELLS , (SPORANGIUM) FREQUENTLY DIFFERS AMONG SPECIES ,MAKING IT OF CONSIDERABLE VALUE IN INDENTIFICATIONS , AS IT MAY LOCATE CENTRAL, TERMINAL OR SUBTERMINAL .

9- SPORE STRUCTURES :

A-THE SPORES OFTEN IS SURROUNDED BY ATHIN DELICATE COVERING CALLED THE EXOSPORIUM .

B-ASPORE COAT LIES BENEATH THE EXOSPORIUM ,IS COMPOSED OF SEVERAL PROTIENS LAYERS AND MAY BE FAIRLY THICK, IT IS IMPERMEABLE AND RESPONSIBLE FOR SPORES RESISTANCES TO CHEMICALS .

C-THE CORTEX, WHICH MAY OCCUPY AS MUCH AS HALF THE SPORE VOLUME , RESISTS BENEATH THE SPORE COAT IT WERE MADE OF PEPTIDO GLYCANS .

D-THE SPORE CELL WALL (OR CORE WALL) IS INSIDES THE CORTEX AND SERROUNDS THE PROTOPLASTS OR CORES .

E- THE CORES HAS THE NORMAL CELL STRUCTURES SUCH AS RIBOSOMES AND ANEUCLOIDES , BUT IT METABOLICALLY INACTIVE.

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :بكر

10 – FACTORS OF ENDOSPORES HEAT RESISTANCES :

1- 15 % OF THE SPORES DRY WEIGHT CONSISTS OF DIPICOLINC ACIDS COMPLEXED WITH CALICIUM IONS WHICH IS LOCATED IN THE CORES. IT HAS LONG BEEN THOUGHT THAT DIPICOLINIC ACIDS WAS DIRECTLY INVOLVED IN SPORES HEAT RESISTANCES .

2- DEHYDERATIONS OF THE PROTOPLASTS VERY IMPORTANT IN HEAT RESISTANCES .

3- THE CORTEX MAY OSMOTICALLY REMOVE WATER FROM PROTOPLASTS ,THERBY PROTECTING IT FROM HEAT AND RADIATIONS DAMAGES.

4-THE SPORES COATS ALSO SEEMS TO PROTECTING AGAINST ENZYMES AND CHEMICALS SUCH AS HYDROGEN PEROXIDES .

11- SPORE FORMATIONS (SPOROGENESIS OR SPORULATIONS):

1-NORMALLY COMMENCES WHEN GROWTH CEASES DUE TO LACK OF NUTRIENTS . IT IS ACOMPLEX PROCESS AND MAY DEVIDED INTO SEVEN STAGES :

1-AN AXIAL FILAMENT OF NUCLEAR MATERIAL FORMS .

2-INWARD FOLDING OF THE CELL MEMBRANE TO ENCLOSE PART OF THE DNA ORODUCE THE FORESPORE SEPTUM .

3-THE MEMBRANE CONTINUES TO GROW TO ENGULFS THE IMMATURE SPORE IN THE SECOND MEMBRANE.

4-CORTEX IS LAID DOWN IN THE SPACE BETWEEN THE TWO MEMBRANES ,AND BOTH CALCIUM AND DIPICOLINIC ACIDS ARE ACCUMULATED .

5-PROTIEN COATS THEN ARE FORMED AROUND THE CORTEX .

6-MATURATIONS OF THE SPORE OCCURS.

7-LYTING ENZYMES DESTROY THE SPORANGIUM RELEASING THE SPORE.

SPORULATION REQIURES ONLY ABOUT TEN HOURS IN BACILLUS MEGATERIUM .

BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE
DEPARTMENT OF CLINICAL PATHOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Youssef Wahhabi

جامعة بنها
كلية الطب البيطرى
قسم الباثولوجيا الإكلينكية
مقدمة فى الباثولوجيا الإكلينكية الحديثة
تحت تحرير وإشراف أ.د خالد محمد رئيس القسم
كتبها ونسقها مساعدباحث/
محمدمحمودمحمديوسف وهيبه
ماجستير البكتيريا والمناعة والفطريات
معهدبحوث الصحة الحيوانية
خاص بمحمدوهيبه

BODY FLUIDS EXAMINATION

FIRST EDITION
COURSE NO. 178

EDITED BY
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PREFACE

THIS IS THE FIREST EDITION OF TUMOR MARKERS. IT IS ATEACHING BOOK WHICH IS DIRECTED AS APOST GRADUATE COURSE. IT IS ORGANIZED BY CLINICAL PATHOLOGY DEPARTMENT FOR POST GRADUATED STUDENT S IN CLINICAL PATHOLOGY OR OTHER ESPACIALISTS.

I ACKNOWLEDGE AND THANK ALL STAFFS IN CLINICAL PATHOLOGY DEPARTMENT FOR THEIR EFFORT AND I HOPE VERY MUCH THAT, IN THIS EDITION, I HAVE MANAGED TO MAINTAIN THE STANDERED OF CONTENT, PRESENTATION AND ACCESSIBILITY.

IN-DEPTH COVERAGE OF THE MORE ESTABLISHED SUBJECTS OF TUMOR MARKERS BOOK INCLUDING : THE CELLULAR AND LIQUID CONSTITUENTS CHARACTERISTICS OF BLOOD , CHARACTERISTICS FEATURES OF NORMAL AND ABNORMAL BLOOD CELL FORMATION AND MATURATION ,

THE STAGES OF LEUCKOCYTE DEVELOPMENT, THE ORDER OF MATURATION AND SERIES AND CHARACTERISTICS OF EACH STAGE WITH VARIATION , OCCURRENCE OF LEUCKEMIA AND ITS CLASSIFICATIONS AND TYPES , THE APPROPIATE SIGNS AND SYMPTOMS FOR THE STAGE OR TYPE OF LEUCKEMIA .

ALSO MOST COMMON TUMOR MARKERS AND ELISA TEST USED FOR ITS DETECTION IN BLOOD ARE WELL COVERED. THESE TOPICS HAVE BECOME IMPORTANT IN TEACHING TUMOR AND ITS BIOMARKERS AS APART OF CLINICAL PATHOLOGY. I BELIEVE I HAVE ACHIEVED HIGH LEVEL OF ACCESSIBILITY AND EASY READING.

KHALID MOHAMED

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WATER METABOLISM

1-WATER CONSTITUTES ABOUT 45-70 % OF TOTAL BODY WEIGHT .

2- BODY FLUIDS ARE DIVIDED IN TWO COMPARTMENTS:

(A)- INTRA CELLULAR (ICF), WHICH WHICH CONSTITUTES 65-75 % OF TOTAL BODY WATER AND 35-45 % TOTAL BODY WEIGHT .

(B)- EXTERA CELLULAR FLUIDS (ECF) CONSTITUTES ABOUT 25 % OF TOTAL BODY WATER AND 15 % OF TOTAL BODY WEIGHT .

3-)- EXTERA CELLULAR FLUIDS (ECF) FOUND IN 3 LOCATIONS :

(A)- INTRA VASCULAR (PLASMA)

(B)-INTRASTITIAL (LYMPH)

(C) – TRANS CELLULAR (IT INCLUDES : CSF ,JOINT FLUIDS ,PERITONEAL PERICARDIAL, PLEURAL AND LIQUID IN INTESTINE).

4-DECREASED TOTAL BODY WATER LEADS TO DEHYDERATION

5-DEHYDERATION IS MANIFESTED BY :

(A)-DECRASED BODY WIEGHT

(B)LOSS OF SKIN ELASTICITY

(C)-DRYNESS OF MUCOUS MEMBRANE

(D)-RETRACTIONOF EYES OF THE EYES

6- ELECTROLYTES AND WATER MOVES FREELY BETWEEN THE ICF AND ECF .

MOVEMENT OF WATER IS DEPENDANT UPON ON HYDROSTATIC PRESSURE AND OSMOTIC PRESUURE OF ECF PROTIENS

7- OSMOTIC PRESSURE OF ANY FLUIDS IS DIRECTLY RELATED TO THE NUMBER OF PARTICLES IN A UNIT OF SOLVENT AND IS MEASURED BY OSMOLE OR MILLIOSMOLE .

8- OSMOLE IS THE MOLECULAR WEIGHT OF THE SUBSTANCE MULTIPLIED BY THE NUMBER OF IONS INTO WHICH IT DISSOCIATES .

9- THE NORMAL OSMOLALITY OF ECF IS ABOUT 300 MOSM /KG

10-ECF CONTAINS SODIUM NA , CHLORIDE CL AND BICARBONATE HCO3

11- ICF CONTAINS K POTTASIUM ,MG MAGNESIUM ,ORGANIC PHOSPHATES AND PROTIEN.

12-SERUM OSMOLALITY CAN BE ESTIMATED BY USING THE CONCETRATION OF NA SODIUM , AND K POTTASIUM AS FOLLOW :

MOSM / KG = 2(NA +K) +BUN/2.8+GLUCOSE/18

13-SERUM OSMOLALITY IS USED AS AMEASURE OF ECF OSMOLALITY .

14-HYPER OSMOLALITY IS FOUND IN CASE OF :
(A)-HYPERNATREMIA
(B)-ACCUMLATION OF ENDOGENOUS SOLUTES SUCH AS GLUCOSE ,KETONE BODIES , AND UREA
15- HYPOOSMOLALITY IS FOUND IN CASE OF HYPONATERMIA
16-OSMORECEPTORS SENSE CHANGES IN OSMOLALITY . WITH HYPER OSMOLALITY (HYPONATREMIA) , OSMORECEPTORS STIMULATES VASOPRESSIN OR ADH SECRETION FROM THE PITUITARY GLAND AND STIMULATE THIRST . THIRST IS STIMULATE BY AS ALITTLE AS

1-2 % CHANGES IN OSMOLALITY .THE END RESULTS IS WATER RETENTION BY THE KIDNEY AND INCREASED WATER INTAKE . INCREASES IN FREE WATER WILL THUS WILL REDUCE SODIUM CONCENTERATION ,OPPOSITECHANGES OCCUR WITH HYPOOSMOLALITY.

17-BARORECEPTORS ARE SENSITIVE TO CHANGES IN EFFECTIVE CIRCULATING VOLUME (ECV) THE ECV IS THAT PART OF THE EXTRACELLULAR FLUID THAT IS IN THE ARTERIAL SYSTEM AND IS EFFECTIVELY PERFUSING THE TISSUES . IT USUALLY VARIES DIRECTLY WITH ECF VOLUME .

WITH HYPOVOLEMIA (DECREASED ECV), BAROECEPTORS STIMULATES THE RENIN-ANGIOTENSIN SYSTEM , THE END RESULT BEING MINERALOCORTICOID (ALDOSTERONE) RELEASE FROM THE ADRENAL CORTEX.

ALDOSTERONE INCREASED ABSORPTION OF NACL AND PROMOTES THE EXCRETION OF POTASSIUM AND HYDROGEN IN THE DISTAL TUBULES OF THE NEPHRON , NACL RETENTION PROMOTES WATER REABSORPTION , THIS CORRECTING THE HYPOVOLEMIA . HYPOVOLEMIA ALSO STIMULATES THIRST (A DECREASE IN ECV OF 7-10 % IS REQUIRED FOR STIMULATION). OPPOSITE CHANGES OCCUR WITH HYPER VOLEMIA

ANTIDIURETIC HORMONE

ADH ACTS ON CHIEF CELLS IN COLLECTING TUBULES , INCREASING PERMEABILITY OF THE TUBULES TO WATER AND UREA . ADH ACTS VIA ADENYLE CYCLASE WHICH OPEN CHANNELS IN THE TUBULES ,CALLED AQUA-PORES , FACILITATING WATER ABSORPTION .ADH IS SECRETED BY HYPOTHALAMUS IN RESPONSE TO OSMOTIC (MAINLY DETERMINED BY NA SODIUM) AND NON OSMOTIC (VOLUME DEPLETION ,NAUSIA ,FEAR,ANXIETY,PAIN,EXERCISE AND DRUGS) STIMULI.

SEVERAL THINGS INHIBIT THE ACTION OF ADH ON RENAL TUBULES AND THUS AFFECT CONCENTRATING ABILITY .

THESE INHIBITORS OF ADH INCLUDE :

(1)-HYPOKALEMIA

(2)-HYPERCALCEMIA

(3)- CORTICOSTEROIDS

(4-)-PROSTAGLANDIN E

IN ANY OF THE ABOVE CONDITIONS URINE CONCENTRATING ABILITY IS IMPAIRED FOR REASONS OTHER THAN PHYSIACL LOSS OF FUNCTIONAL NEPHRONS .

(1- SODIUM)

SYNONUM : SERUM SODIUM NA +

SPECIMEN : SERUM OR PLASMA (HEPARINE) I ML .

REFERENCE VALUE : 136-145 MEQ/LITRE ; SI UNITS : 136-145 MMOL/LITRE

METHODS : ION-SELECTIVE ELECTRODE ,SPECTROPHOTOMETER ,FLAMEPHOTOMETER .

DESCRIPTION :

1-SODIUM IS THE MAIN CATION OF EXTERACELLULAR FLUIDS WHERE AHALF OF TOTAL BODY SODIUM IS PRESENT .MOST OF THE REST SODIUM IS PRESENT IN BONES IN STABLE FORM .

2- SODIUM HAS TWO MAIN FUNCTIONS :

A-REGULATION OF ACID BASE BALANCE

B-PLAY AN IMPORTANT ROLE IN REGULATION OF OSMOTIC PRESSURE

3-THE QUANTITY OF SODIUM IS CONTROLLED BY SODIUM INTAKE AND LOSS.

4- HORMONAL CONTROL :

A-IT IS PEFORMED BY ALDOSTERONE HORMONE ,WHICH WHICH SECRETED BY ADRENAL S GLANDS : ITS ACTION IS PERFORMED ON THE RENAL TUBULES WHERE IT CONTROLS EXCRETION AND REABSORPTION OF SODIUM .

B-MOST OF SODIUM PRESENTED TO RENAL TUBULES IS REABSORBED (APPROXIMATELY 90 %) UNDER CONTROL OF ALDOSTERONE HORMONE .

C-IF THERE IS AN EXCESS OF BODY SODIUM ,ALDOSTERONE SECRETION IS REDUCED AND SODIUM IS EXCRETED THROUGH KIDNEYS :BUT IF THE SODIUM IS REDUCED ,ALDOSTERONE SECRETION AND PRODUCTION IS INCREASED AND SODIUM IS ALMOST COMPLETELY REABSORPED .

N.B SODIUM REABSORPTION REQIURES AN EQUIVALENT PASSAGE OF HYDROGEN OR PUTASSIUM IONS IN THE OPPOSITE DIRECTION .

D-EXCRETION OF SODIUM :

IT IS DONE THROUGH :

1-MAINLY KIDNEYS THROUGH URINE.

2-SWEAT .

3-SECRETIONS OF DIGESTIVE TRACTS .

4-THROUGH STOOL.

HYPONATREMIA

DECREASE IN PLASMA SODIUM CONCENTRATION IS CALLED HYPONATERMIA
IT OCCURS MOST FREQUENTLY BECOUSE OF EXCESSIVE SODIUM LOSS THROUGH THE FOLLOWING ROUTS :

1-DIGESTIVE TRACT :

A-DIARRHEA b – VOMITING

2-URINARY TRACT :

A-RENAL DISEASES RENAL FAILURE b– USING OF DIURETICS-

D- DIABETES MELLITUS AND HYPER GLYCEMIA .

3-EXCESSIVE SWEATING WITHOUT ADEQUATE SODIUM INTAKE .

4- EXCESSIVE EXUDATION FROM BURNS OR OPEN WOUNDS WHEN SODIUM INTAKE IS INDEQUATE .

5-ADRENOCORTICAL INSUFFICIENCY (ADDISON S DISEASE).

6-LIPEMIA IN WHICH LIPIDS DISPLACES ASIGNIFICANT VOLUME OF LIQUID PHASE OF PLASMA MAY RESULT IN ALOW PLASMA SODIUM ,SIMPLY BECOUSE SMALLER VOLUME OF LIQUID PHASE .

6-CONGESTIVE HEART FAILURE .

7-ADMINISTERATION OF HYPOTONIC SOLUTION SUCH AS 5% DEXTROSE .

9-METABOLIC ACIDOSIS .

10-INSUFFICIENT INTAKE .

HYPERNATREMIA

1-HYPERNATREMIA IS ARARE CONDITION .

2-IT OCCURS WHEN THERE IS LOSS OF FLUIDS CONTAIN ASMALL AMOUNT OF SODIUM AS IN DIABETES INSPIDUS .

3-EXCESSIVE SODIUM INTAKE WITH LIMITED LIQIUD INTAKE .

4-REDUCED GLOMERULAR FILTERATION RATE AS IN RENAL FAILURE.

5-HYPER ALDOSTERONISM .

2- POTASSIUM

SYNONUM : SERUM K+

SPECIMEN: SERUM OR PLASMA HEPARINE I ML .

REFERENCE VALUE : 3.9 – 5.8 % MEQ/L .

METHODS : ION –SELECTIVE ELERCTRODE, FLAMOPHOTOMETER, SPECTROPHOTOMETER .

DESCRIPTION :

1-POTASSIUM IS THE MAN CATIONS IN THE CELLS OR INTRACELLULAR

2- FUNCTION OF POTASSIUM ARE :

A-CONTROL OF MUSCULAR ACTIVITY

B- CONTROL NEURAL EXCITABILITY

3-EXCRETION OF POTASSIUM IS DONE THROUGH:

A-KIDNEY

B-STOOL

C-SWEAT

D- DIGESTIVE FLUIDS

4-ALDOSTERONE FACILITATES EXCRETION OF POTASSIUM AS IT INCREASE SODIUM REABSORPTION BY PROMOTING EXCHANGE OF SODIUM FOR POTASSIUM .

POTASSIUM IS PRESENT IN MORE HIGH CONCENTERATION INTRACELLULAR ,SO THAT NECROSIS OF TISSUES OR DESTRUCTION OF RBCS CAUSES HIGH SERUM POTASSIUM LEVEL .

HYPO KALEMIA :

1-DIARRHEA AND VOMITTING ESPECIALLY WHEN REPLACEMENT FLUIDS IS POTASSIUM (K+)FREE .
2-IATROGENIC AS IN CASE OF PROLONGED ADMINSTERATION OF MINERALOCORTICOID.
3-HYPERALDOSTERONISM
4-ALKALOSIS ,POTASSIUM DECREASES BECOUSES IT ENTERS INSIDE THE CELLS EXCHANGING WITH HYDROGEN IONS AND ALSO INCREASE EXCRETION THROUGH URINE.
5-BICARBONATE THERAPY
6-DIURETIC TREATMENT
7-RENAL TUBULAR ACIDOSIS
8-PROLONGED ANOREXIA
9-EXCESSIVE SWAETING
10-CHRONIC RENAL FAILURE
11-INSULIN TREATMENT IN KETOACIDOTIC DIABETES (WILL CARRY POTASSIUM INTO THE CELLS ALONG WITH GLUCOSE).

HYPER KALEMIA

1-ACUTE RENAL FAILURE WHERE THERE IS ANURIA OR OLIGOURIA

2-HYPOADRENOCORTICISM (ADDISONS DISEASE)

3-USING OF POTASSIUM SPARCING DIURETICS

4-INCREASE INTAKE (AS POTASSIUM CHLORIDE (KCL) USED IN CONGESTIVE HEART FAILURE)

5-ACIDOSIS

6-DIFFUSE CELL DEATH SECOUNDARY TO SHOCK ,CIRCULATORY STASIS CHEMOTHERAPY

7-FACTITIOUS (ERRORS DURING COLLECTION OF SAMPLE):

a-HEMOLYSIS –b-DELAYED SEPARATION OF CLOTTED BLOOD (DUE TO POTASSIUM LEAKAGE) –c- COLLECTION IN POTASSIUM HEPARINE.

CYSTATIN C

CYSTATIN C

SPECIMEN: SERUM OR PLASMA (HEPARIN)
METHODOLOGY: IMMUNOASSAY
REFERENCE VALUE: 0, 53 - 0, 95 mg /L

Description

Description :

1- CYSTATIN C IS ACYSTIENE PROTIENASE INHIBITOR .

2-ASMALL MOLECULE THAT IS PRODUCED BY NUCLEATED CELLS .

3-SERUM CYSTATIN C IS FILTERED OUT OF BLOOD BY THE KIDNEYS AND THAT SERVES AS AMEASURE OF KIDNEYS FUNCTION.

4- ITS MOLECULAR MASS ALLOWS IT TO BE FREELY FILTERED BY THE GLOMERULAR MEMBRANE IN THE KIDNEYS.

5-SINCE IT IS FORMED AT ACOSTANT RATE AND FREELY FILTERED BY THE HEALTHY KIDNEYS.

6-CYSTATIN C IS AGOOD MARKER OF RENAL FUNCTION.

7-SERUM CONCENTERATIONS OF CYSTATIN C ARE ALMOST TOTALLY DEPENDANT ON GLOMERULAR FILTERATION RATE (GFR) .

8-AREDUCTION IN (GFR) CAUSES ARISE IN THE CONCENTERATION OF CYSTATIN C.

9-CYSTATIN C IS ABETTER MARKER OF THE GLOMERULAR FILTERATION RATE AND HENCE OF KIDNEY FUNCTION THAN CREATININE WHICH WAS THE MOST COMMONLY USED MEASURE OF KIDNEY FUNCTION.

10- THE LEVELS OF CYSTATIN C ARE INDEPENDENT OF WEIGHT AND HEIGHT ,MUSCLE MASS,AGE, (OVER AYEAR OF AGE) ,SEX,AND FACTORS WHICH HAVE BEEN DOMENSTERATED TO AFFECT CREATININE VALUES,IN ADDITION,ARISE IN CREATININE DOES NOT BECOME EVIDENT UNTIL (THE GFR) HAS FALLEN BY APPROXIMATELY 50%.

11- MEASUTEMENTS CAN BE MADE AND INTERPRETED FROM ASINGLE RANDOM SAMPLE.

12- CYSTATIN C MAY IS USED AS AN ALTERNATIVE TO CREATININE AND CREATININE CLEARANCE TO SCREEN FOR AND MONITOR KIDNEY DYSFUNCTION IN THOSE WITH KNOWN OR SUSPECTED KIDNEY DISEASES.

13- IT MAY BE ESPECIALLY USEFUL IN THOSE CASES WHERE CREATININE MEASUREMENT IS NOT APPROPRIATE: FOR INSTANCE IN THOSE WHO HAVE LIVER CIRRHOSIS, ARE VERY OBESE, ARE MALNOURISHED OR HAVE AREDUCED MUSCLE MASS.

14- MEASURING CYSTATIN C MAY ALSO IS USEFUL IN THE EARLY DETECTION OF KIDNEY DISEASE WHEN OTHER PARAMETERS MIGHT STILL BE NORMAL, ESPECIALLY IN THE ELDERLY.

THANK YOU

BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE
DEPARTMENT OF CLINICAL PATHOLOGY

Written and presented by

Doctor Mohammad Mahmoud Mohammad Youssef Wahhabi

جامعة بنها
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قسم الباثولوجيا الإكلينكية
مقدمة فى الباثولوجيا الإكلينكية الحديثة
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خاص بمحمدوهيبه

THE TOPIC TODAY

Renal failure

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
28109091800516/2004578801966

Renal failure is a state in which the body's metabolism is disturbed as a consequence of dysfunction of the kidneys, characterized by numerous manifestations, which are attributable to the imbalance between the rate of production of various chemical substances by the body and their rates of the excretion by the kidneys.

Kidneys become unable to perform its regulator role of the electrolytes, acid-base balance and excretion of waste products.

(Syndrome of renal insufficiency)

There are two characteristics syndromes of renal failure:-

1-acute renal failure.

2-chronic renal failure.

They Are So Two Distinct , And Each One Consider Alone .

1-Acute Renal Failure :

Definition :

It Means Acute Suppression Of Kidney Filtration Resulting In Rapidly Increasing Azotemia , With Or Without Oliguria .

Acute Renal Failure Can Be Divided Into Two Phases :

1-Oilguric Phase .

2-Diuretic Phase .

1-Oilguric Phase Which Is Characterized By :

A-Sever Decrease Of Urine Volume .

B-Increased Level Of Urea And Creatinine .

C-Decreased Serum Na+ And Hyper Kalemia.

D-Sediments Shows Protein , Red Cells ,Epithelial Cells And Casts.

2-Diuretic Phase :

Begins 2 Weeks Post Onset , Gradual Increase Of Urine Volume Indicating Nephrons Recovery (Continues For 7-10 Days).

Causes Of Acute Renal Failure :-

1-Pre Renal Causes
(Vascular Diseases).
2-Renal Causes .
3-Post Renal Causes .

1-Pre Renal Causes (Vascular Diseases):-

Sudden Vasoconstriction Of Renal Arteries In The Most Important Cause Of Acute Renal Failure.

Vasoconstriction Occurs In The Following Condition:-

A-Sudden Decrease In Blood Volume Due To Hemorrhage.

B-Cardiac Failure:

C-The Presence Of The Large Quantities Of Free HB And Myoglobin.

D-Certain Completion Of Pregnancy As Eclampsia ,Concealed Accidentals Hemorrhage And Abortion.

C-Postoperative.

:++++2-Renal Causes : It Includes

A-Glomular Diseases:

*-Acute Glomerulonephritis Caused By Salmonella ,Shigella And Toxoplasma.

*-Pyelonephritis With Rapidly Necrosis Caused By Coryne Bacterium Renale.

*-Systemic Lupus Erythematosis.

*-Membranous Glomerulonephritis.

B-Vascular Diseases:

*-Acute Bilateral Renal Artery Obstruction As Caused Thrombosis And Or Embolism.

*-Disseminated Intravascular Coagulation (DIC).

*-Thrombotic Thrombocytopenic Purpura (TTP).

C-Interstitial And Tubular Diseases:

*-Acute Interstitial Nephritis Caused By Leptospira.

*-Acute Tubular Necrosis :This May Occur Toxic Or Ischaemic.

3-Post Renal Causes :

***-Acute Urinary Tract Obstruction Occur Due To :**

A-Stones In The Pelvis Or Ureters Or Urethera Or Urinary Bladder.

B-Prostatic Tumor Or Stressful Hypertrophy .

C-Blockage Of The Tubules By Casts Or Crystals Of Uric Acid Or Sulphonamides

Laboratory Findings :

1-Passage Of Small Amount Of Dark Urine (Oliguria).

2- Changes In Blood.

3- Hematological Changes .

1-Passage Of Small Amount Of Dark Urine (Oliguria)

A-Urine Contains Protein , Blood And Cellular Debris .

B-Specific Gravity Is Variable (Usually High)

2- Changes In Blood:

A-Retention And Increased Level Of Urea And Creatinine ,Potassium ,Magnesium ,Phosphate other Anions.

B-Metabolic Acidosis.

C-The Blood Calcium Is Some Lowered Secondarily To The Raised Blood Phosphate.

D-A considerable Increase In Extracellular Water.

E-Decrease In The Serum Concentration Of Sodium And Chloride.

3- Hematological Changes Include :

A-Rapidly Developing Anemia (Normocytic Normochromic Anemia) And Is Accentuated By Diluting Effect Of The Increased Blood Volume Secondarily To Water Retention .

B-Hemolytic Type Of Anemia Occurs To Due To Uremia.

C- Neutrophilic Leuckocytosis Is Also Present.

JUST ASIMPLE NOTICE OF DOCTOR MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA
ON SOME MASTER AND PHILOSPHY DOCTOR THESIS
E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :
1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM
IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
28109091800516 - 2004578801966

FULL PROFFESORS - SEVEN-LASIK.pdf

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THE TOPIC TODAY
CHRONIC RENAL FAILURE

DOCTOR / MOHAMMED MAHMOUD MOHAMMED YOUSEF WEHABA
28109091800516/2004578801966

DEFINITION :

IT MEANS CHRONIC INSUFFICIENCY OF RENAL EXCRETORY AND REGULATING FUNCTIONS LEADING TO UREMIA

CHRONIC RENAL FAILURE RESULTS FROM PROGRESSIVE DECREASE IN GLOMERULAR FILTERATION AND THE LOSS OF TUBULAR FUNCTIONS.

CAUSES :

1-CHRONIC DESTRUCTIVE RENAL DUSEASES.

2-CHRONIC RENAL VASICULAR DISEASES.

3-OSBSTRUCTION TO THE URINE OUTFLOW.

4-CONGENITAL LESIONS .

5-METABOLIC CONDITIONS.

1-CHRONIC DESTRUCTIVE RENAL DISEASES INCLUDES:

1-CHRONIC PYELONEPHRITIS
2-CHRONIC GLOMERULONEPHRITIS
3-SYSTEMIC LUPUS ERTHEMATOSIS
4-BILATERAL RENAL TUBERCULOSIS
5-RADIATION NEPHRITIS

2-CHRONIC RENAL VASCULAR DISEASES :

1-MALIGENANT HYPER TENSION.
2-RENAL VEIN THROMBOSIS

3-OBSTERUCTION TO THE URINE OUTFLOW

1-THIS IS MANIFESTED BY BILATERAL HYDERONEPHROSIS AND OCCURS SECONDERILY TO URETHERAL OBSTERUCTION FOLLOWING :

1-ASTRICTURE

2-PROSTATIC ENLARGEMENT.

3-CARCINOMA OF THE CERVIX ,WHICH MAY ALSO OCCLUDE BOTH URETERS

4-CONGENITAL LESIONS AS :

1-POLY CYSTIC DISEASE
2-CERTAIN TUBULAR DEFECT MAY EVENTUALLY LEAD TO SEVER RENAL FAILURE

: - 5-METABOLIC CONDITIONS

1-HYPER CALCEMIA ,HYPOKALEMIA , AMYLOIDOSIS , GOUT
2-DIABETES MELLITUS (DIABETIC NEPHROPATHY)
ALL MAY LEADS TO SERIOUS RENAL DYSFUNCTION ESPECIALLY IF THEY PERSIST FOR LONG DURATION.

LABORATORY FINDINGS : INCLUDES :

1-DISTURBANCE IN WATER METABOLISM.

2-HYPONATREMIA.

3-MILD RETENTION OF THE POTASSIUM.

4-DISTURBANCES IN ACID BASE BALANCE .

5-DISTURBANCES IN CALCIUM AND PHOSPHORUS METABOLISM.

6-IMPAIRED GLUCOSE TOLERANCE AND INCREASED INSULIN RESISTANCE IS COMMON.

7-RETENTION OF WASTE PRODUCTS : UREA ,URIC ACIDS , CREATININE.

8-HYPERTENSION .

9-HEMATOLOGICAL EFFECTS

LABORATORY FINDINGS :

1-DISTURBANCE IN WATER METABOLISM : CLEARLY SHOWS :

***THE INABILITY OF THE FAILING KIDNEY TO CONCENTERATE THE URINE ADEQUATELY LEADS TO MODERATE POLYURIA ,WHICH IS CONSIDERED EARLY SIGNS IF RENAL FAILURE THE POLYURIA ALSO OCCURS DUE TO OSMOTIC OSMOTIC DIURESIS**

***POLYURIA IS ESPECIALLY MARKED IN CHRONIC PYELONEPHRITIS AS THE CONCENTRATING PART OF MEDULLA IS AFFECTED EARLY.**

***AMILD DEGREE OF DEHYDERATION OCCURS AND IS MANIFESTED BY THIRST.**

4-DISTURBANCES I ACIDBASE BALANCE :

PROGRESSIVE RENAL FAILURE IS ACCOMPANIED BY AMETABOLIC ACIDOSIS DUE TO RELATIVE FAILURE OF ACIDIFICATION OF URINE DUE TO IMPAIRENT IN AMMONIA PRODUCTION ,AND THE ABILITY TO SECRETE HYDROGEN ION (H+) IS IMPAIRED.

5-DISTURBANCES IN CALCIUM AND PHOSPHOROUS :METABOLISM

IN CHRONIC RENAL DISEASE:THERE IS APERSISTANT RETENTION OF PHOSPHATE AND SERUM PHOSPHATE RAISED

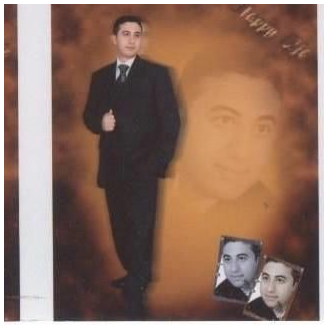
THE SERUM CALCIUM IS SOMEWHAT REDUCED AS AN INVERSE RESPONSE TO THE RAISED SERUM PHOSPHATE AND ALSO DUE TO IMPAIRED INTESTINAL ABSORPTION OF CALCIUM (BECOUSE OF VITAMINE D RESISTANCE AND DEFICIENCY)

SECOUNDARY PARATHYROID HYPERPLASIA MAY OCCUR ,THE PARA THORMONE MOBILIZES CALICIUM AND PHOSPHATE FROM THE BONES THEREFORE ,OSTEOMALACIA (RENAL RICKETS)OCCURRED.

9-HEMATOLOGICAL EFFECTS AND PATHOLOGICAL CHANGES:

SEVERE NORMOCYTIC NORMOCHROMIC ANEMIA IS COMMON IN THE LATER STAGES OF RENAL FAILURE . ANEMIA IS ATTRIBUTED TO THE IMPAIRED PRODUCTION OF ERYTHROPOITIEN,WHICH IS NORMALLY FORMED IN THE KIDNEY.

PURPURA ,WHICH IS PROBABLY DUE TO AQUALITATIVE DEFICIENCY IN THE PLATELETS.



Some of Important bacterial diseases

Written and presented by

Doctor

Mohammad Mahmoud Mohammad Youssef wehaba

Assistant Researher

Master of bacteriology ,immunology,mycology

Animal health research institue

Some of Important bacterial diseases

Streptococcus	pneumonia , pharyngitis , cellulitis
Staphylococcus	abscess of skin and other organs
Bacillus anthraces	anthrax
Clostridium	tetanus , gas gangrene , botulism

Coryne bacterium	diphtheria
Listeria	meningitis
Actinomyces	actinomycosis

Nocardia	nocardiosis
Neisseria	gonorrhea , meningitis
Haemophilus	meningitis
Bortedella	whooping cough
Legionella	pneumonia
Brucella	brucellosis
Francisella	tularemia

Pasteurella	cellulitis
Yersinia	plaque
Escherichia coli	urinary tract infection , diarrhea
Enterobacter	urinary tract infection
Serratia	pneumonia
Klebsiella	urinary tract infection
Salmonella	enterocolitis , typhoid fever

Shigella	**enterocolitis**
Proteus	**urinary tract infection**
Campylobacter	**enterocolitis**
Helicobacter	**gastritis , peptic ulcer**
Vibrio	**cholera**
Pseudomonas	**urinary tract infection , pneumonia**
Bacteroids	**peritonitis**

Mycobacterium tuberculosis , leprosy
Rickettssia (rocky mountain spotted fever , typhus , Q fever)
Chlamydia urethritis , trachoma , psittacosis
Treponema syphilis
Borrelia lyme disease
Leptospira leptospirosis
Mycoplasma pneumonia

THANK YOU

التصنيفات للبكتيريا المهمة طبيا

جامعة بنها

كلية الطب البيطرى

قسم البكتيريا والمناعة والفطريات

كتبهاونسقها مساعدباحث محمدمحمودمحمديوسف وهيبه

ماجيستير البكتيريا والمناعة والفطريات

Classification of medically important bacteria

1- RIGID, THICK- WALLED CELLS	2- FLEXIBL, THIN WALLED CELLS	3- WALL LESS – CELLS
	Leptospira	**Mycoplasma**

1- RIGID, THICK- WALLED CELLS

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)	B- NON-FREE-LIVING BACTERIA (OBLIGATE INTERACELLAR BACTERIA (as Rickettsia and Chlamydia)

A- FREE- LIVING BACTERIA (EXTERA CELLULAR BACTERIA)		
1- Gram positive bacteria (stained appear blue colour)	2- Gram negative bacteria (not stained just Avery little appear red or pink colour)	3-Acid fast bacteria e.g T.B Mycobacterium

<table>
<tr><td colspan="3">1- Gram positive bacteria (stained appear blue colour)</td><td colspan="2">2- Gram negative bacteria (not stained just Avery little appear red or pink colour)</td></tr>
<tr><td>a- Cocci
Staph
Strept</td><td colspan="2">b- Bacilli include : Spore forming and Non Spore Forming</td><td rowspan="4">a- Cocci like Nisseria</td><td rowspan="4">b- Bacilli include : Aerobic (Psudomonas) and Facultative Anaerobic (Straight – Curved) and Anaerobic bacteria (Bacteroides)</td></tr>
<tr><td rowspan="3"></td><td>Spore forming</td><td>Non Spore forming</td></tr>
<tr><td>Aerobic bacteria like Bacillus Anthracis
Anaerobic bacteria like Clostridia</td><td>non Filamentous like
Coryn bacteria,
Listeria and Filamentous Actinomyces</td></tr>
<tr><td></td><td></td></tr>
</table>

Facultative anaerobic

Straight			Curved
Respiratory bacteria Haemophilus	Zoonotic bacteria- Brucella , Pasteruella , Yersenia	Enteric bacteria E.coli , Salmonella, Shigella ,Klebsiella, Proteus	Campylobacter , Vibriocholera ,Hilicobacter

بعض التحصينات الإجبارية للأمراض الوبائية والكشف عنها داخل القطر المصرى
(كتبها ونسقها
دمحمدمحمودمحمديوسف وهيبه
بعد إرسالها المركزى) -

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

المرض المشتبه به	نوع العينة المراد العزل منها	المادة الحافظة	طريقة النقل المناسبة ودرجة الحرارة المطلوبة	المدة المسموح بها حتى الوصول إلى معهد الدقى
١-الحمى القلاعية (FMD)	حويصلة كاملة-بثرات- سائل الحويصلة	Glycerin buffer	فى ثلاجة نقل العينات محتوية على أكياس ثلج خاصة (4C) بحيث تكون كل عينة فى كيس منفصل	٢٤ ساعة
٢-حمى الوادى المتصدع (RFV)	دم على مانع تجلط –أجنة مجهضة (كبد-طحال-غدة ليمفاوية)	Universal transport medium	كما سبق	٢٤ ساعة
٣-الجلد العقدى (LSD)	عقدة جلدية – عقدة ليمفاوية – طحال	Transport buffer	كما سبق	٢٤ ساعة
٤-طاعون المجترات الصغيرة (PPR)	دم على مانع تجلط أثناء إرتفاع درجة الحرارة –بثرات من الفم – مسحات من العين والأنف والفم – أحشاء (الرئة غدد ليمفاوية – الأمعاء الطحال)	EDTA Heparin	كما سبق	٤-٦ساعات

٥-التسمم الدموى Pasteurella multocida	مسحات أنفية – أحشاء داخلية – دم –سيرم		كماسبق	٤-٦ساعات
٦-حمى الثلاث أيام Bovine ephemeral fever virus Rhabdoviridae	دم على مانع تجلط – غدة ليمفاوية - طحال	Preservative media PBS	كما سبق	٢٤ ساعة

٧- الإلتهاب الشعبى البقرى (IBR)	مسحات أنفية-غدة ليمفاوية-جزء من الرئة – جزء من القصبة الهوائية	Preservative media PBS	كما سبق	٢٤ساعة
٨-الإسهال المعدى (BVD)	مسحات أنفية-مسحات شرجية-سيرم-غدة ليمفاوية – أحشاءداخلية	Preservative media PBS	كما سبق	٢٤ساعة
٩-أمراض العجول الرضيعة pneumoenteritis,parainfleunza ,corona viruses,may be mixed with salmonella , mycoplasma, E,coli	عينات براز-سيرم-أحشاءداخلية-مسحات أنفية ودم	Preservative media PBS	كما سبق	٢٤ساعة

١٠-التسمم المعوى (Enterotoxaemia) Cholesteridial infections especially in sheep and goat farms need co vac 8	الأمعاءالدقيقة (مربوطة ولا يقل طولها عن ١٥ سم) –فص كامل من الكبد به الإصابة – كلية كاملة		كما سبق	٤-٦ساعات
١١-التفحم العضلى والأوديما الخبيثة Black Leg (black quarter) Malignant Edema Closteridium Choviae	العضلة المصابة المتفحمة – سائل إرتشاحى من مكان الإصابة		كما سبق	٤-٦ساعات
١٢-المرض الأسود Black Closteridium (Disease) Choviae	فص كامل من الكبد به إصابة –الأمعاء الدقيقة		كما سبق	٤-٦ساعات

١٣-باسيلارى هيموجلوبين يوريا (بول أحمر) (Bacillary Hemoglobinuria) Clostridium novyi type D	فص كامل من الكبد به الإصابة		كما سبق	٤-٦ساعات
١٤-تعفن الحافر (Foot Rot) Fusobacterium necrophorum	مسحات من الحافر المصاب بعد تنظيفه جيدا من الأنسجة الميتة	المسحات على ناقل (Cary-Blair Medium)	كما سبق	٤-٦ساعات
١٥- التسمم الغذائى	-الأعلاف (لا تقل العينة عن ٥٠-١٠٠جرام) -السيلاج (لا تقل العينة عن ٥٠-١٠٠ جرام)- المياة (لا تقل عن ٢٠٠ مل)		كما سبق	لا تزيد عن ١٢ ساعة بطريقة الحفظ المذكورة

ملحوظة هامة : البروسيلاوالإسبريجلوسيس يسببان عاصفة من الإجهاضات ومن الأمراض التى يجب تبليغ جميع السلطات الأمنية الغذائية ويكتب ويرفع بها تقرير مبين وتم نسيانها من المختصين العاليين

يجب الإفادة عن الموقف التحصينى لتلك الأمراض عند إرسال أى عينات للفحص

وكيل المعهد للتشخيص المعملى وصحة الأغذية

تحريرا فى : / /

يعتمد

أ.د

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

جامعة بنها

كلية الطب البيطرى

قسم البكتيريا والمناعة والفطريات

كتبهاونسقها مساعدباحث محمدمحمودمحمديوسف وهيبه

ماجيستير البكتيريا والمناعة والفطريات

معهد بحوث الصحة الحيوانية

مركز البحوث الزراعية

وزارة الزراعة

(أعوذبالله من الشيطان الرجيم)

(بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ)

(وَقُلِ ٱعۡمَلُواْ فَسَيَرَى ٱللَّهُ عَمَلَكُمۡ وَرَسُولُهُۥ وَٱلۡمُؤۡمِنُونَۖ وَسَتُرَدُّونَ إِلَىٰ عَٰلِمِ ٱلۡغَيۡبِ وَٱلشَّهَٰدَةِ فَيُنَبِّئُكُم بِمَا كُنتُمۡ تَعۡمَلُونَ)

(التوبة - ١٠٥)

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS

FAMILY: ENTEROBACTERIACEAE

THE DEFINITION OF ENTEROBACTERIACEA:

Intestinal bacteria describe large biochemically and antigenically related groups of organisms

FAMILY: ENTEROBACTERIACEAE

1-Some of them are pathogenic potential to man, animals, insects and plants.

2- It contains about 4 tribes, 12 genera on the basis of DNA relatedness.

3-Generally the members of family enterobacteriaceae is composed of gram negative rods which may be motile by peritrichous flagella or non-motile.

4- They are non -spore forming and not acid fast.

5- Most of them are non capsulated but some have capsules or microcapsules (slime layer).

6- Enterobacteriaceae members grow on the presence and the absence of free oxygen because facultative anaerobes have the presence of respiratory and fermentative metabolisms.

5- They grow well on ordinary media.

6- They are similar in the morphological and cultural characters.

7-Moreover all species contain the enterobacterial common antigen.

8- Biochemically they produce acid and gas due to the fermentation of glucose or different types of carbohydrates.

9- All species are catalase positive .

10-Reduce nitrate to nitrite.

11- Oxidase negative .

12- According to their ability to ferment lactose they were differentiated biochemically into two divisions :

1 - Lactose fermenting enterobacterial genera.

2-Non lactose fermenting enterobacterial genera .

This classification depends on the cultivation of the species of family enterobacteriaceae on one of the following differential selective media :

1-MacConkey agar (lactose-bile neutral red agar)

2-S.S media (salmonella- shigella agar)

3- Desoxycholate cirate agar

4- Hegkton media

Different genera of Enterobacteriaceae according to their ability to ferment lactose : Lactose fermenter and Non Lactose +(Reacted Fundamental Environment) fermenter

Lactose fermenter :

Genus	Habitate
E.coli	Intestine
Enteobacter	Intestine , water
Citrobacter	Intestine, water , soil
Yersenia	Intestine, water
Serratia	Intestine, water , ,soil

Non- lactose fermenter :

Genus	Habitate
Shigella	Intestine ,water, soil
Salmonella	Intestine , water , soil
Hafnia	Intestine, water,soil
Proteus	Intestine, water , soil
Morganella	As Proteus

FUCULTATIVE ANAEROBIC GRAM NEGATIVE RODS
FAMILY: ENTEROBACTERIACEAE

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.THANK YOU .

GENUS: ESHCERICHIA
SPECIES: ESCERICHIA COLI (E.Coli)
Synonym: BACTERIUM COLI (E.Coli)

Definition: Escherichia coli is a normal inhabitant of the lower part of intestine of all warm blooded animals.

It is rarely present in stomach and in few numbers in the anterior part of intestine. Carnivores and omnivores usually harbor E.coli in greater number than herbivore.

Under certain conditions of stress and environmental factors may undergo to be potential pathogenic causing sever diseased conditions.

Specific diseases caused by enteropathogenic E.coli in different animal species:

Species	Disease	Symptoms
Childrens	Coli septicemia	**Diarrhea , death**
Newly born animals	Colibacillosis, white or calf scour	**Septicemia and rapid death**

Microscopic characters :

E.coli is coccobacilli to long rods (.6 x1.3M), arranged singly or in short chains especially in mucoid colonies where it had a slime layer of polysaccharides . it is motile and non – sporulated .

Cultural characters :

(Nutrient broth-Nutrient agar-Blood agar-MacConkey agar)

-In nutrient broth growth is rapid , gives regular turbidity within 12-18 hours at 37 C with heavy powdery sediment after 24 hours .

-On nutrient agar growth

1-smooth surface colonies occurs after 24 hours,

-The colonies are grayish in color , circular , convex 2-3mm in diameter .

 2- mucoid colonies appear due to the presence of slime layer and larger in size than smooth one .

-On blood agar the surface colonies are larger some are hemolytic and others are non-hemolytic

-On macConkey agar it gives round pink colonies, medium sized opaque colonies i.e lactose fermenter

(Viability And Resistance) :

-It may survive for weeks or months in contaminated water, dwellings in the animal houses in the presence of mucous or feces . Some strains resist dryness.

-It is susceptible to physical and chemical agents. E.coli is killed by heating at (55-60 C) for 30 minutes, within 10 minutes by autoclaving.

The Antigenic Structures Of E.coli include three antigens : 1-somatic antigens 2- capsular antigens 3- flagellar antigens . Detected by serological diagnosis

1-Somatic antigens (146 O- antigens)
2-Capsular antigens (91 K –antigens)
3- Flagellar antigens (49 H- antigens) :

1-Somatic Antigens : (146 O- antigens) :

E. coli strains have 146 O-antigens in their body . Each strain carries only one type of O-antigen . They are composed chemically of polysaccharide–phospholipids –protein complex. The O –antigens are designated by numbers as O1, O2, O3,O4 ,O5 – O 145 etc .

The O – antigens are heat stables.

2- Capsular antigens (91 K –antigens) :

These 91 k-antigens occur as a slime layer or microcapsule on most enteropathogenic E.coli strains.

Chemically they are composed of poly saccharides . k-antigens prevent agglutination between O-antigens and their specific homologous antisera.

The K – antigens are differentiated into

3 types according to their thermo stability as follows :

L-antigen : heat labile

A- antigen : heat stable

B- antigen : lipopoly saccharide destroyed by autoclaving

The K-antigen is very important in serotyping of E.coli as well as in the mechanism of pathogenesis as K 88 and K 99 .

These K 88 and K 99 mediate adhesion of the organism to the microvilli of the intestinal epithelial cells and enhance colonization .

3-Flagellar antigen (49 H-antigens) : There are 49 H-antigens which is composed of protein . frequently it is poorly developed .

It has weak role in serotyping of E.coli.

-Serotyping Identification :

For serotyping of E. coli , identification of O and K antigens is applied by slide agglutination test to determine its antigenic structures as O20/B6 , O55/B5 , O88/B4 , O111/B4 .

Toxins of E.coli (E.coli products) :

1-Enterotoxin 2- colicins

1-Enterotoxin:

This substances is produced by entero pathogenic strains of E. coli , and causes diarrhea in man and animals . Injection of E .coli enterotoxins into the ligated intestinal loops of rabbits induced swelling , oedema and accumulation of exudate in the injected loop. The enterotoxins are not produced by all E.coli strains but only the enteropathogenic.

E.coli enterotoxin is composed of two fractions:

a- Heat stable fraction.
b-Heat labile fraction.

a-Heat stable fraction : it is exteracellular in the culture . it is resistant to heating for 60 C but destroyed by autoclaving .
b- Heat labile fraction: it is inactivating by heating at 60 C for 30 minutes it is antigenic and occurred within bacterial cells . it could be neutralized by specific antisera .

2- Colicins :

It is E.coli products and composed chemically of protien . it has bactericidal activitiy . colicins productions is very important in differentiation of E.coli strains into :

Colicins positive E.coli.

Colicins negative E.coli.

Laboratory diagnosis:

- The isolation of E.coli may be of no value as E.coli are normal inhabitant of the intestinal tract, unless other seven factors are considered including:

1-Detailed history of the case, symptoms and lesions with differential bacteriological diagnosis on the other causative agents of digestive disorders .

2-Purity of the growth of E.coli on the primary cultures note the growth of lactose fermenting colonies on MacConkey agar .

3-Site of isolation from various organs especially from the parenchymatous organs as liver , spleen , mesenteric lymph nodes and lung tissues indicating colibacillosis.

4- Biochemically identification of isolated suspected E.coli the suspected E.coli colonies as lactose fermenter on macConkey media can be tested biochemically And must be differentiated from the other lactose – fermenter species by the following reactions (Indol , Methyle Red , VP , Citrate , Urea Hydrolysis , H2S, Motility , KCN) (Voges proskauer)(potassium cyanide)

E.coli are motile and Indol and Methyl Red Positives Only .

5-Serotyping of E. coli if the isolating colonies, are biochemically typical of E.coli, Serotyping must be performed using polyvalent O, K anti-sera to determine the antigenic structure of the isolated strain of E.coli.

6-Final diagnosis depends mainly upon the isolation of E.coli from various fresh organs in case of septicemia of the same antigenic structure.

7-It is very essential that the same serotype of E.coli must be isolated from feces or intestinal content as well as the internal organs of the same case .

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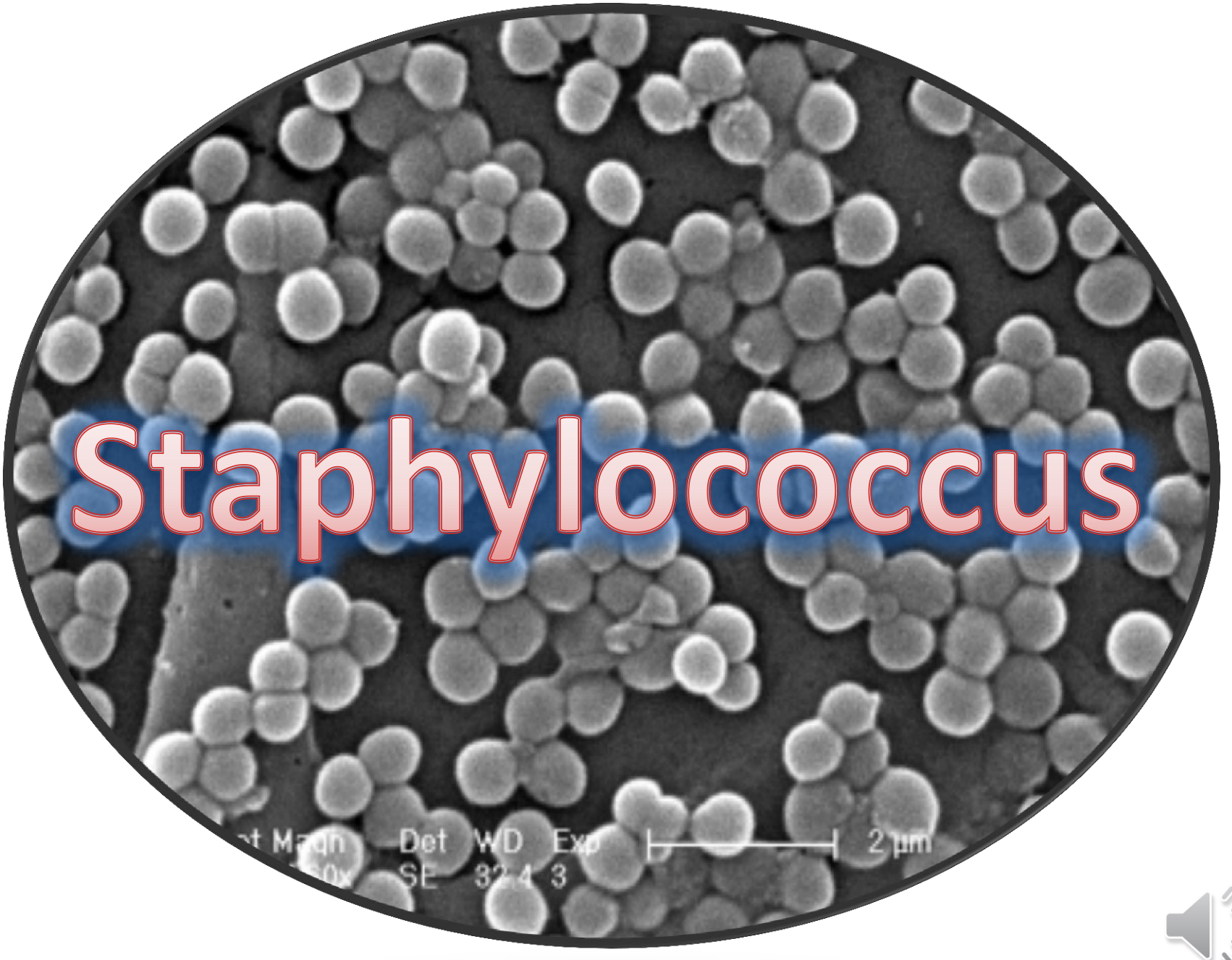
Staphylococcus
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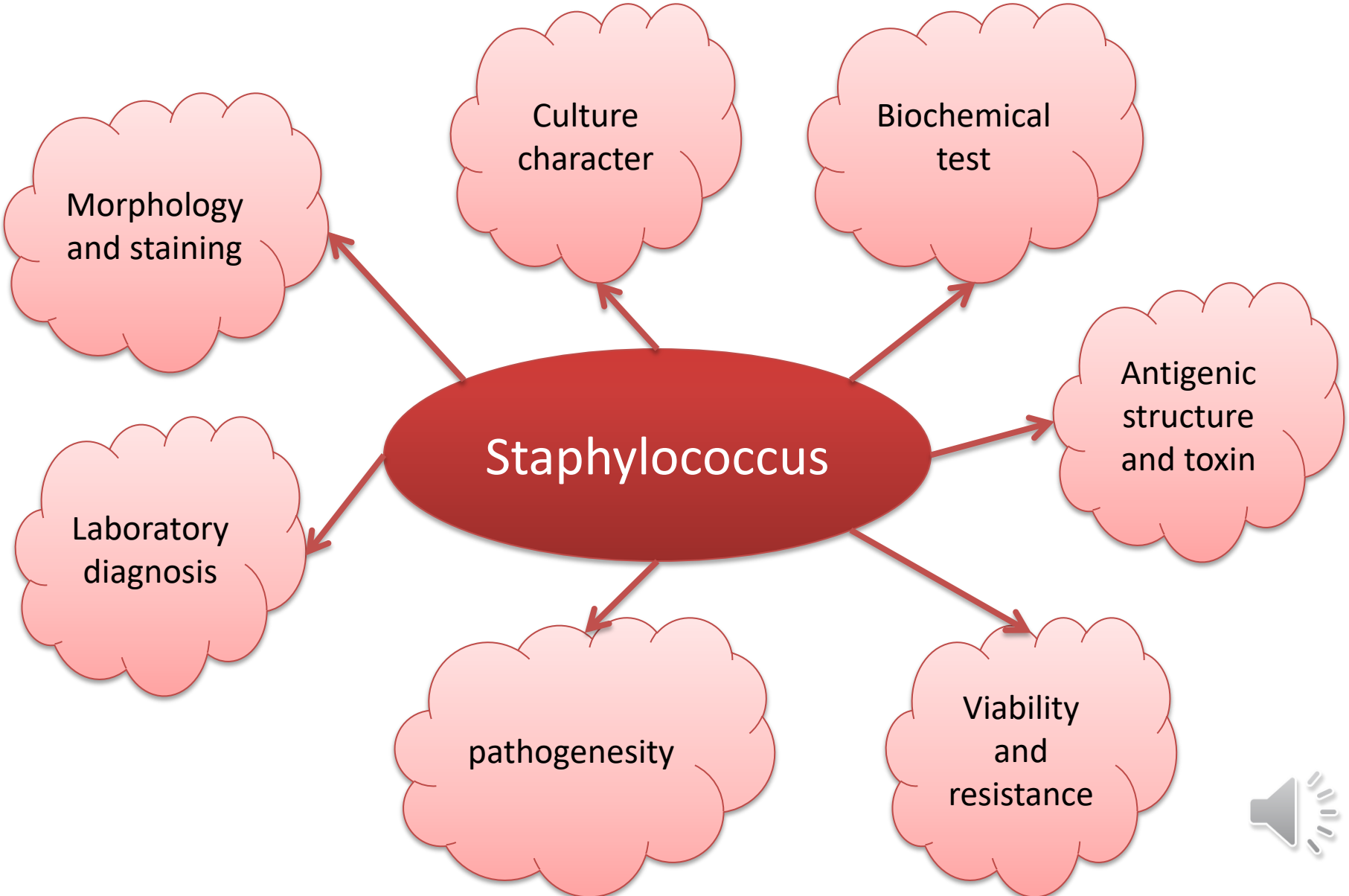
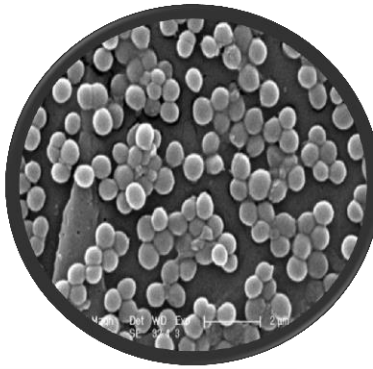
2 µm

What must I know in microbe?



Morphological character and staining

Gram reaction	gram-positive cocci violet in color
shape	spherical or ovoid cells arranged in grapes like cluster
motility	non motile
capsule	non capsulated
Spore formation	non sporulated



culture character

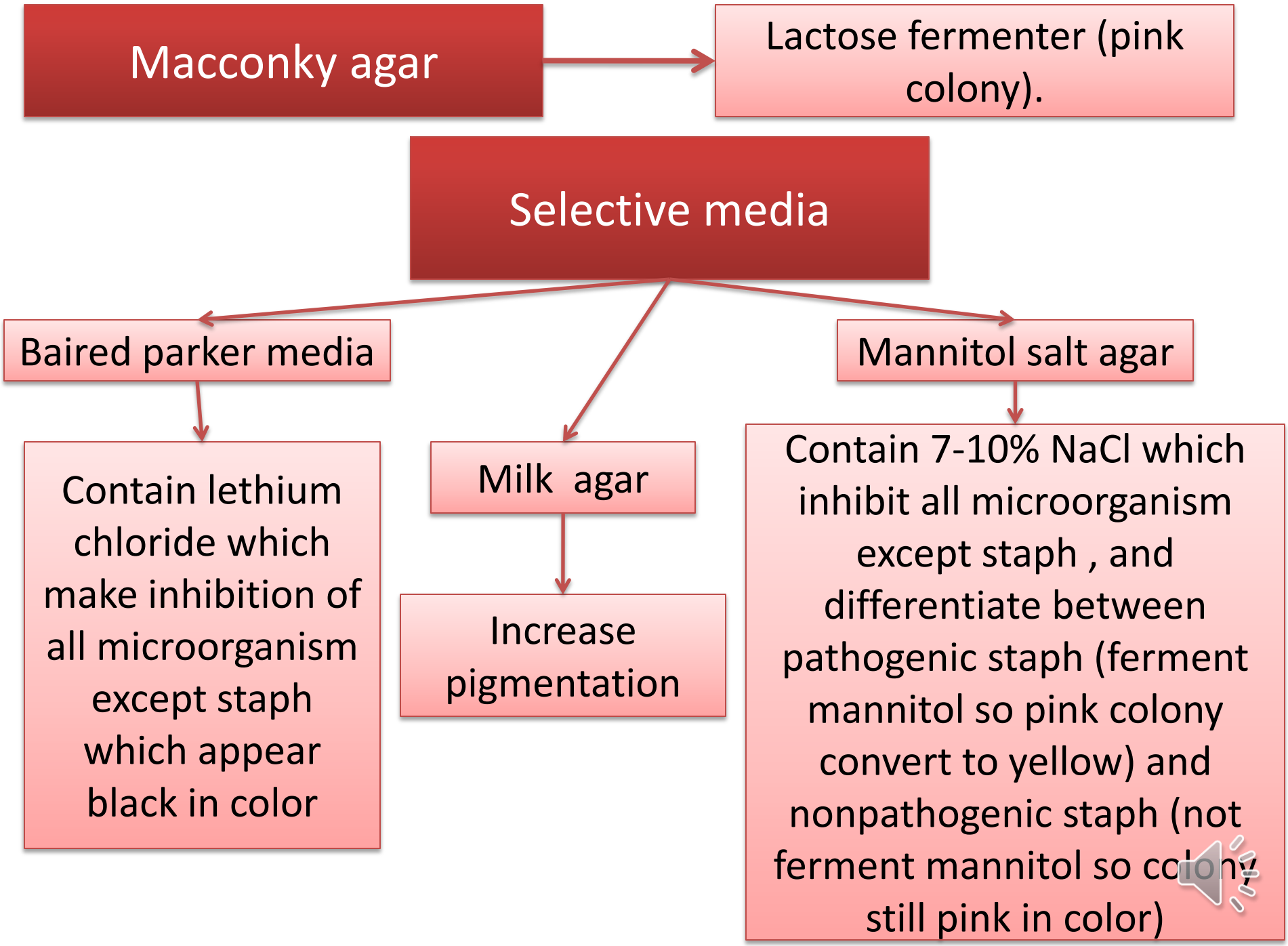
Grow of microbe on : media need to

oxygen	aerobic and facultative anaerobic
Temperature	37 celsius degree
PH	7.2
Incubation period	After 24 hours

culture character

media

Blood agar	are round, convex smooth, golden or white or lemon in color, with zone of complete haemollysis
Nutrient broth	uniform turbidity is present with powdery sediment and no pigments.
Nutrient agar	Small smooth endopigmented colony (colony only pigmented)


Macconky agar
Lactose fermenter (pink colony).
Selective media
Baired parker media
Mannitol salt agar
Contain lethium chloride which make inhibition of all microorganism except staph which appear black in color
Milk agar
Increase pigmentation
Contain 7-10% NaCl which inhibit all microorganism except staph , and differentiate between pathogenic staph (ferment mannitol so pink colony convert to yellow) and nonpathogenic staph (not ferment mannitol so colony still pink in color)

Biochemical test

Sugar fermentation test	as lactose, glucose, mannitol giving acid only
indol	-Ve
Methyle red(M.R)	+Ve
Vogus proskauer(V.P)	+Ve
Coagulase test	with pathogenic strain +Ve (clotting serum)
Catalase test	+Ve

Antigenic character and Toxin

Staph.aureus produces the following exo-toxins and enzymes:

1-coagulase enzyme:

Coagulated citrated plasma and it is used to differentiate between pathogenic and non-pathogenic staphylococcus strains, as in this picture which show postitive coagulase at the left (*S.aureus*) and negative at the right(non pathogenic strain)

2-leucocidin :

It is a substance which destroys leucocytes, haematoblasts of the bone marrow.

3-fibrinolysin: dissolve blood clot.

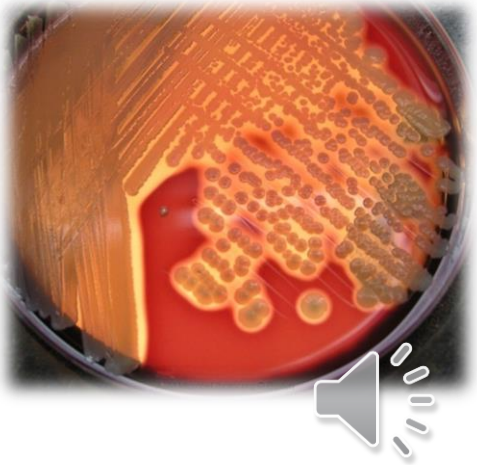
4-**hyalourinidase enzyme**: (spreading factor) it breaks down the hyalouronic acid.

5-haemolysins:

A substance which causes haemolysis to R.B.Cs and this organism has 3 types of haemolysins:

.

α-haemolysin:	- β-Haemolysin:	γ-Haemolysin:
causing haemolysis of rabbbit R.B.Cs ,sheep R.B.Cs.	- causing haemolysis of sheep R.B.cs ,weak on rabbit	causing haemolysis of rabbit R.B.Cs ,sheep R.B.Cs,human R.B.Cs



6-Enterotoxin:

It causes food poisoning in man. This toxin did not destroyed by 100 c for 30 minute while N/100 HCL destroys the enterotoxin.

7-lethal toxin:

It causes death in rabbits, cats, guinea pigs and mouse.

8-Dermonecrotoxin:

It causes necrosis of skin; it has been demonstrated experimentally in the rabbit and occurs in natural infection in the pastular dermatitis in dogs

Viability and resistance

-it is the most resistant of cocci.
-killed by heating at (60-80 C) for 30 minutes.
-killed by gentian violet at 1/25,000 after 30 sec.
-killed by 10%formaldhyde in 10 minutes
-it is very sensitive to the antibiotics such as penicillin, streptomycin and neomycin

•there are penicillin resistant strains (MRSA) (methicillin resistant staphylococcus aureus)

The penicillin normally affected on staphylococcus by B-lactam which affected on ability of bacteria to produce mucopeptide which is the main component of cell wall (essential structure) while resistant strain have ability to produce B-lactamase enzyme which make destruct of penicillin B-lactam so no effect of penicillin antibiotic with this modefied strain (resistant strain)

pathogenesity

Staphylococcus aureus usually associated with the following:

-Suppurative wound infection in man and animals.

- Mastitis in animals.

- pastular dermatitis in dogs.

-Food poisoning in man.

-Botryomycosis in horse.

Laboratory diagnosis

pus Sample:

Direct smears: from pus stained with gram stain to demonstrate gram positive cocci arranged in grape like clusters.

Isolation of the organism on specific media as mannitol salt agar.

Detection of the enterotoxin in case of food poisoning.

Thank you for attention

Clostridia

Species:

Clostridium tetani
Clostridium botulinum
Clostridium perferingens (CL.welchii)
Clostridium septicum **and** ***Clostridium chauvoei***
Clostridium novyi (CL.oedematiens)

Definition:

This genus are spore forming rods which are anaerobic, some of them are pathigenic for man and animals.

The Clostridial group grows only in the absence of free oxygen (O_2) due to its lack of catalase and peroxidase enzymes which are necessary to split H_2O_2as end metabolites where H_2O_2 are toxic and harmful to the clostridium group. While free oxygen itself was not harmful to clostridium

General characters of Clostridial group :

Size and shape:

The members of clostridial group are bacilli with variable dimensions measuring (0.4 – 1.2µ × 3-8µ) in length. They are usually plump in shape with round ends, but some are long and slender rods.

Sporulation:

All of members are sporulated with rhe exception of *Cl.perfringens* spores have variable position and which may be central, sub-terminal or terminal.

Motility:

Most species are motile with pertrichous flegella except *Clostridium welchii* which is non motile.

Staining:

All members are gram positive mainly in young cultures.

Capsule:

Most members are non capsulated except *Cl. perferingens*.

Toxin production:

All pathogenic species give one or more types of exotoxins.

CLOSTRIDIUM WELCHII (CLOSTRIDIUM PERFRINGENS):

This group of organism are most commonly distributed in the intestinal tract of man and animals, there are 6 toxogenic typed (A,B,C,D,E,F) and differentiated according to the types of toxin produced. It causes different disease in man and animals.

Morphology:

It is gram positive and straight with parallel sides and rounded ends, spores are rarely seen. The organism are non motile and it is capsulated.

Cultural characters:

Clostridium welchii is not strict anaerobe; its surface colonies are circular, convex, semi translucent, smooth and white and entire edge, there is no tendency of spreading over the surface of the medium.

On horse blood agar: many strains produce double zones of haemolysis due to production of two toxins (theta and alpha) and absence of haemolysis is a characteristics of food poisoning strains of *Clostridium welchii* .

On heated blood agar: few strains produce evidence of slight proteolytic activity.

On egg-yolk agar: the organism produces opalescence which is inhibited by the specific *Cl. Welchii* antitoxin (Nagler reaction).

Nagler reaction (lecithinase activity)

Several clostridia are able to produce opalescence in both human serum and egg yolk media, due to the production of lecithinase which cause visible egg yolk media, due to the production of lecithinase which cause visible precipitates in these media.

The reaction is demonstrated with alpha (α) toxin of *Cl. Welchii* and is specifically inhibited by *Cl.welchii* anti toxin.

Nagler reaction has also been used for presumptive identification of *Cl.welchii* in direct plate culture when one half of the egg yolk medium inoculated with 2 or 3 drops of stander antitoxin and allowed to dry.

The whole plate is then inoculated with *Cl.welchii* starting with the half free from the antitoxin. On the half containing no antitoxin, *Cl. welchii* colonies shows surrounding zone of opacity i.e. Nagler reaction, while colonies, shows no change on the antitoxin half.

The Nagler reaction of the Clostridium members:

Organism produce opalescence	**Opalescence inhibited by *Cl. welchii* (alpha antitoxin)**
Cl. welchii (A-F)	+
Cl. Botulinum	-
Cl.oedematiens Type: A B D	 - - -

Biochemical Reactions:

In cooked meat media, the growth is evident in afew hours and the meat particles changes to pink, i.e. due to its saccharolytic activity.

All types ferment glucose, maltose, fructose, sucrose and liquefy gelatin.

Litmus milk is coagulated and acid and gas is produced giving stormy fermentation as in picture of test tube. H_2S is positive, indol negative, MR negative, VP negative and catalase negative.

Resistance and viability :

The vegetative form of the organism is not resistant to heat or chemicals, in spore form it is quite heat resistant, not being killed for 10 minute at 120 °C.

Toxins of *Clostridium welchii*:

The types of *Cl. Welchii* differ in the combination of various toxic and enzymic factors they produce. Several of these factors have haemolytic , lethal or necrotizing properties and others have enzymatic activity against biological substrate . the types of *Cl. Welchii* can be differentiated on the bases of four major lethal toxins (alpha, beta, epsilon and iota).beside to

these four major toxins there are 8 minor toxins (theta ,gamma, mu, nu, delta, kappa, leta and lambda).

Major toxin:

Alpha toxin: (α)

It is produced by all types of *Cl. welchii*, it is lethal and necrotizing , this toxin is an enzyme lecithinase C, and in presence of calcium ions it splits lipoprotein opalescence . The reaction can be inhibited by specific antiserum.

Beta toxin :(ß)

Produced by types B and C it is lethal and necrotizing.

Epsilon toxin:

It is produced by types B and D strains as a protoxin and therefore it needs a proteolytic enzyme e.g. pepsin or trypsin to be activated it is lethal and necrotizing, its activity depends also beside the proteolytic enzyme in alkaline medium .

Iota toxin:

Only type E produces this toxin as a protoxin so it needs pre-enzyme to be activated.

Minor toxins of *Cl. Welchii* :

Theta toxin:

It is an oxygen labile haemolysin , it is produced by all strains of *Cl. welchii* from A-F. It is lethal and hemolytic toxin.

Gamma toxin: it is lethal toxin.

Delta toxin:

It is lethal toxin and haemolytic for red cells of sheep, goat ,pigs and cattle.

Kappa toxin: it is collagenase which attacks native collagen and may cause softening of muscle connective tissues.

Mu toxin : It is a hyalurinidase

Nu toxin: It is deoxyribonuclease enzyme

Distribution of major toxins in different types of *Cl. welchii* and disease produced.

Cl. welchii types	Major toxin produced	Disease produced
A	Alpha	Gas gangrene in man and animals
B	Alpha, beta, epsilon	Lamb dysentery
C	Alpha, beta	Struck in sheep
D	Alpha, epsilon	Pulpy kidney disease in sheep
E	Alpha, iota	Enterotoxemia in calves and lambs

Typing of *Cl.welchii* strains

Clostridium welchii strains could be typed either by:

Neutralization test :

It is test of *Cl.welchii* was prepared and part (0.3) is put in 6 test tubes. The first tube left as control and antiserum (0.1) of types A.B.C.D and E was added to other test tubes respectively.

The tubes incubated at 37°C for 1hr. then 0.1 ml of each tube injected intra-venous in white mice and the deaths recorded after 24 hr.

The groups of mice which remain alive represent the type of *Cl. welchii*. i.e. if group pf mice injected each toxin (B) remain alive so type of *Cl. welchii* is type B and the first group must be died.

N.B. In neutralization test , antitoxin must be added to the toxin and not the reverse.

Dermonectrotic test:

Toxin of *Cl. welchii* was also prepared and antisera of types A, B, C,D and E are added. Then 0.1 ml of the toxin as it is or from the mixture (toxin –antitoxin) is injected intradermally on the back of guinea pigs after

removing the hair then left 24 hours and the result indicated by the presence of necrosis at the site of injections.

Nagler reaction or lecithinase activity:

Laboratory diagnosis of Cl. welchii:

Isolation and identification of the causative organism.

lecithinase activity or Nagler reaction on egg yolk media.

Demonstration of the type of strain and the toxin present in the intestinal content,

Identification and typing was applied by neutralization test using specific antitoxic sera by animal inoculation.

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Isolation and identification of the causative organism.

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Demonstration of the type of strain and the toxin present in the intestinal content,

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Salmonella

- Comprise a large group of serotypes (2700)

- World wide & infect a wide variety of hosts

As man , animals & poultry

- Salmonella are typical intestinal pathogens

& contaminate the environment by the organism in the feces and transfer infection to other

Some species are host spcific

species	group	host	disease
S.Paratyphi A	A	Man	Paratyphoid A fever
S.Paratyphi B	B	Man	Paratyphoid B fever
S.Abortus equi		Equine	Equine abortion & infertility
S.Abortus ovis		Ovines	Ovine abortion
S.cholera suis	C	swine	Piglet typhus Infectious enteritis
S.typhi	D	man	Human typhoid fever
S.gallinarum		poultry	Pullorum disease

salmonella

Genus : Salmonella

Species : S. typhi

S. typhimurium

S. enteriditis

S. gallinarum-pullorum

S. dublin

S. infantis

S. derby

S. agona

S. panama

S. heidlberg

morphology

- Cocco-bacilli
- Gram -ve
- Medium sized rods
- Actively motile expect S.gallinarum and pullorum
- Many species developed fimbria
- Non spore forming
- Non capsulated

Cultural characters

- **Faculatative anearobes . Temrpature 37C PH 7.2 incubation time 48 hours**
- **Grow in ordinary media**
- **In nutrient broth** **nutrient agar**

Turbidity , without Pellicle formation

round colonies, smooth, convex with grayish colour

S. gallinarum & S. abortus ovis

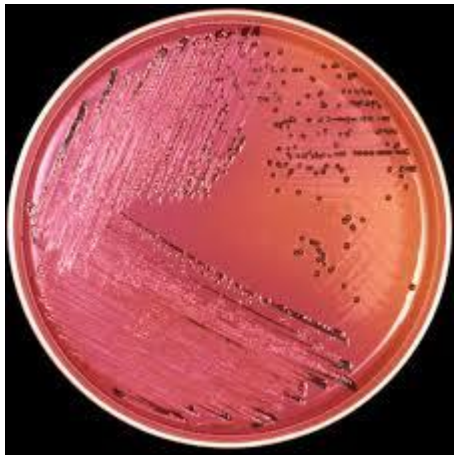
- **Onto blood agar ⟶ the colonies relatively large , non haemolytic**
- **Onto MacConkey agar pale in colour (non lactose fermenter)**

Selective media of Salmonella

There two selective media of salmonella

1- XLD (Xylose Lysin Deoxscholte) agar : colonies appear pink calour with black center due to salmonella produce H2S

2- S.S agar (Salmonella Shigella Agar) : give yellow coloniesn



Enrichment media

There is a more complex problem for salmonella isolation due to their presence in the intestinal tract

Examples :

- **Tetrathionate brilliant green broth**
- **Selenite F broth**

by growing the fecal samples or intestinal contents on the enriched media for 18 -24 hours to inhibit the growth of the other

Biochemical reactions

- Lactose -ve
- Indole -ve
- MR +ve
- VP -ve
- Citrate +ve
- Urease -ve
- H2S +ve

Viability & resistance

- Salmonellae easily killed at 60ºC for 20 min.

- Quickly destroyed by the common chemical disinfectant

- **Identification of salmonella species depends mainly on the serotyping according to**

Kaufman & White scheme of the antigenic structure of salmonella

Antigenic structures

- **Somatic antigens O-antigen**

- **Present in the cell wall**

- **Lipopolysaccharide-protein complex**

- **Designated by arabic numbers from 1 – 65**

- **The majority of salmonallae posses more than one of somatic antigens (3-4) on their**

- **Flagellar & fimbrial antigens (H-antigens)**

- **Present in motile strains except S.gallinarum**

- **- protein in nature**
- **- salmonella have 2 phases of flagellar antigens**

- **Phase 1 H**	**phase 2 H**
- **Identified by alpha-are**	**20 H antigens**

- **Virulent antigen (Vi – antigen)**
- **Present in freshly isolates of S.typhi**
- **Vi – antigen lost by subculturing**

Examples of salmonella serotypes

	S. paratyphi A	S. paratyphi B
group :	A	B
O-antigen :	1,2,12	1,4,5,12
H-antigen		
phase 1 :	a	b
phase 2 :	-	1,2
	monophasic	Diphasic

Morphological character and staining of E.Coli

ساعات معتمدة ملف التسمم الغذائى دكتوراة مادةالبكتيريا

1-gram reaction gram negative coccobacilli – violet in color

2-shape coccobacilli to long rods singly or short chains

3-motility by peritrichous flagella

4-capsule - capsulated with slime layer

5-spore formation – non sporulated

Cultural character needs:

Oxygen : facultative anaerobic

Temperature : 37 C cellius degree

PH : 7.2

Incubation periods : after 24 hours

Selective media :

1-Eosin methylene blue agar (EMB) : green metallic colony

2-violet red bile agar (VBRA) : pink colony

Lactose fermentation : positive , H2S : negative , citrate utilization : negative , , oxidase : negative , catalase : positive , potassium cyanide : negative , vogus prouskour : negative , indol : positive , methyle red : positive . urea test : negative .

TOXINS OF ESCHERICHIA COLI :

1-ENTERO TOXINS

2-COLICINS

3-VEROTOXINS (SHIGA TOXINS): RESPONSIBLE ON BLOODY DIARRHEA

SURVIVAL AND RESISTANCE :

KILLED BY ANTIBIOTICS

PATHOGENESITY :

E.COLI CLASSIFIED IN TO SIX PATHOTYPES :

ENTEROPATHOGENIC E.C (EPEC)

ENTEROINVASIVE E.COLI (EIEC)

ENTEROHEMOLYTIC E.COLI (EHEC)

ENTEROTOXIGENIC E.COLI (ETEC)

ENTERO AGGREGATIVE E.COLI (EAgEC)

ENTEROUROPATHOGENIC E.COL I (EUEC)

PATHOGENICITY OF ESCHERICHIA COLI ON THE HUMAN AND ANIMALS AND POULTRY :

1-HUMAN FOOD POISONING WITH DIARRHEA , INFLAMMATION OF URINARY BLADDER

2-COWS AND BUFFALOS ONE OF MASTITIC CAUSE OR CAUSES OF MASTITIES AND COLIBACILLOSIS ESPECIALY IN NEWLY BORN CALVES

3-POULTRY CAUSING COLIBACILLOSIS AND CRD CHRONIC RESPIRATORY DISEASES WHICH ARE AMIXTURE BETWEEN E.COLI AND MYCOPLASMA

DIFFER BETWEEN FECAL FORM AND NON FECAL FORM :

BY USING EIJKMAN TEST :

IT IS DEPENDANT ON THE ABILITY OF BACTERIA TO GROW ON MACCONKEY AGAR 44 C CELLIUS DEGREE :

IF IT IS ABLE TO GROW ON THIS TEMPRATURE IT IS FECAL FORM E.COLI

IF IS NOT ABLE TO GROW ON THIS DEGREE IT IS NOT FECAL FORM E.COLI

LABORATORY DIAGNOSIS :

SAMPLING :

1-MASTITIS : MILK SAMPLE

2-DIARRHEA : FECES

3- INFLAMMATIONS OF URINARY TRACT : URINE

4-SAMPLE FROM VOMITION IF PRESENT

Benha University
Faculty of Veterinary Medicine
Bacteriology, Immunology, Mycology Department

Occurrence of food borne pathogen in some types of soft cheese in local market

A Thesis Submitted to
Faculty of Veterinary Medicine
Benha University

By

Mohammed Mahmoud Mohammed Yousef Weheba
(BVSc., Kafer Elsheikh University, 2003) For the degree of M.Sc.
(Bacteriology, Immunology, Mycology)

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Tanta branch

بِسۡمِ ٱللَّهِ ٱلرَّحۡمَٰنِ ٱلرَّحِيمِ

(قالوا لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم)

Acknowledgement

First of all, I wish to express my deep thanks to Allah Almighty who has endowed his help and blessings on me to have the present work achieved in such condition.

I wish to express my gratitude and thanks to Prof. Ashraf A wad Abdel El tawab, professor of Bacteriology, Immunology, Mycology, Faculty of Veterinary Medicine, Benha University for his guidance, criticism, continuous encouragement and helpful advice during this work.

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Thanks, you all for your patience and support

VITA

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He works at animal health research institute, Tanta branch, Connected To The Main Institute In The Dokki -Giza -Cairo Under Super Visions Of Agricultural Researches Center Under Laws Of Universities Learning And Researches Qualifications.

Dedications

My Father
My Mother
My Sister
My Brother
My Uncles
My Family

Always all support religious forever.

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1. INTERODUCTION

Food safety is considered one of the most common urgent matters in the food industry worldwide. Spoilage of the food products with foodborne pathogens receive a special concern among food producers, investigators and customers. Consequently, producing a safe food represents one of the most imperative urgencies in the food processing (Friedman *et al.*, 2002; Mohamed *et al.*, 2013).

The outbreaks of foodborne illnesses caused by *E. coli* have been studied previously in developing countries after ingestion of milk products such as old-style cheese which is considered the major source of various types of pathogenic bacteria (Elhaida and Mohammed, 2013).

E. coli **is one of the major significant bacteria, which has a bad effect on both human and animal species. Thus, this type of bacteria can deteriorate the milk particularly raw milk and other milk products as a result of poor hygienic measures (Lara *et al.*, 2016; Garbage *et al.*, 2016).**

Prevention of foodborne illness is a global concern that affects all aspects of food production, including dairy. Dairy cattle are known reservoirs for zoonotic bacteria and milk and milk products have been implicated in outbreaks of foodborne illness (Wells *et al.,* 1991; Zhao *et al.,* 1995; Wallace, 1999). These outbreaks are generally caused by improperly pasteurized products, post pasteurization contamination, or by raw milk and raw milk products.

pathogenic *Escherichia coli* are also present in dairy production environments and have been associated with milk and milk products (Ryser, 2001; Watseka and Kinema, 2003). Although milk can be contaminated with bacterial pathogens via shedding in the udder, these zoonotic pathogens are not commonly associated with mastitis. Fecal contamination is considered to be the most common route of bulk tank milk contamination with these organisms.

Escherichia coli **live commensally in the gastrointestinal tract of most mammals, including humans, without causing disease. However, a small fraction of *E. coli* are human pathogens and have been implicated in foodborne illness with increasing frequency over the last 2 decades. Ingestion of enteropathogenic *E. coli* (EPEC) can result in a mild gastrointestinal disease that is relatively self-limiting but a subset of the EPEC, can a serious potentially fatal illness (O'Brien and Kipper, 1998). The most common human clinical EPEC isolated in the United States is O157:H7 (Canavalia *et al.,* 2001; Watseka and Kinema, 2003).**

E. coli **is classified into six pathotypes: enteroaggregative, enterohemorrhagic Shiga toxin-producing *E. coli* (STEC), enteron invasive, enteropathogenic, enterotoxigenic, and diffuse adherent (Jafari *et al.*, 2012). lethal STEC which cause severe illnesses were also detected (Boutin *et al.*, 2007).**

Previous studies indicated that STEC represents one of the most significant pathotypes which lead to foodborne illnesses compared with other types *E. coli* (Brett *et al.*, 2003; Kaufmann *et al.*, 2006). In human, the pathogenic effect of STEC nearly due to its ability to produce certain types of cytotoxins for example Shiga toxins (*stx1*), and intimin (*eae A*) virulent genes (Slaney *et al.*, 2009 and Assupol *et al.*, 2015).

Cheesemaking is a major industry worldwide, and much of it is still practiced on a relatively small scale with only basic hygiene practices (Kostas *et al.*, 2010; Dias *et al.*, 2012).

Among the dairy products, cheese is considered one of the most public sources of vital nutrients (*e.g.* vitamins, minerals and proteins) which represent the main part of healthy food (López-Expositor *et al.*, 2012). In Egypt. Moreover, Kariesh cheese is a prevalent type of cheese which contains a higher protein content with a small amount of fats (Hamad, 2015).

Nevertheless, cheese is considered a safe food for human being, sometimes its deterioration by various types of foodborne pathogens may take place. Enteropathogenic *E. coli* (EPEC) are considered the most common bacteria isolated from cheese. EPEC also cause high morbidity and mortality rates among young and old people (Kostas *et al.*, 2010).

Kariesh cheese are the most popular soft cheese in Egyptian cities and Arabian countries, similar to Domiati (Brown, 2004; Abd-Hamid, 2012; Hegazi *et al.,* 2012). It is an acid coagulated fresh cheese, made from skimmed buffalo's or cow's milk or a mixture of both with soft composition, white curd and slightly salty (Francois *et al.,* 2004; Abou-Donia , 2008), but mostly produced from skimmed buffaloes' milk and its manufacturing depends on acid coagulation by the action of rennet & lactic acid bacteria

Escherichia coli **is commonly present in the intestinal tracts of humans and animals, and its detection in processed foods is an indication of fecal matter contamination. It also indicates the inadequate controls of the environment of the cheeses processing units, the personals hygiene's of the food handlers, and the poor qualities of the finished products (Kostas *et al.*, 2010; Dias *et al.*, 2012).**

Because the occurrence of an Outbreaks of a foodborne disease is caused by Enteropathogenic *E. coli* and is associated with the consumptions of soft ripened cheese (Maurier *et al.*, 1973), the presence of these microorganisms in cheese has taken on a major significance.

Shiga toxins–producing *E. coli* (STEC), also known as serotoxins-producing *E. coli* (VTEC) or Enterohaemorrhagic *E. coli* (EHEC), have been known as a group of highly pathogenic *E. coli* strains producing one or more Shiga toxins (Monaghan *et al.*, 2011).

STEC represent a hazardous public health problem worldwide causing various human gastrointestinal tract diseases, including watery or bloody diarrhea and might develop a life-threatening disease, such as hemorrhagic colitis (HC), Thrombotic Thrombocytopenic Purpura (TTP) and Hemolytic Uremic Syndrome (HUS) (Pennington, 2010).

STEC strains produce two powerful phages encoded cytotoxins causing tissue damage in humans and animals, called Shiga toxins or serotoxins (*Stx1/VT1* and *Stx2/VT2*), which are the common features and main virulence factors of STEC and are directly correlated with human pathogenicity. (Lindgren *et al.*, 1993).

Stx1 **is one of the most powerful toxins, and the toxin producing strains are usually associated with more severe infections (Monies *et al.*, 2004 and Giles, 2007).**

STEC serotypes including O157:H7 STEC and non-O157. The O157:H7 STEC strain is considered one of the most important of all known food-borne pathogens because of the severity of associated illnesses produced by it and also, the apparent low infective dose (of less than 10 cells) (Bach *et al.*, 2002, Blanco *et al.*, 2003).

The most common, non-O157 strains associated with disease in humans including O26, O91, O103, O111, O128, O113 and O145 are responsible for up to 20 to 50% of all STEC infections (Johnson *et al.*, 2006).

There is a lack of information concerning the stress responses of many non

O157:H7 STEC causing human illness under stress conditions as most of these studies only focused on O157:H7 (Allen *et al*. 2008; Dong and Schell horn 2009; Vanaja *et al*. 2010). *eae* Agene it an intimin secretion gene that enhance attachment and colonization of *Escherichia coli* in the intestinal epithelium leading to sever cellular destruction and severe infections and sever symptoms like profuse diarrhea and ulcerative colitis (*Ibrahim et al.*, 2019*).*

Damiati cheese is one of soft pickled cheese and makes up about 75% of the cheese produced and consumed due to its nutritional value, convenience and have rich taste, strong sharp flavor as well as smooth body and texture.

Tallaga cheese is the most popular local type of packaged or unpackaged soft cheeses by all socioeconomic classes in Egypt due to its nutritional values, conveniences and good tastes, clean pleasant creamy low salty taste with a spreadable mellow soft body. It is a product closely related to Domiati cheese and mainly ready for consumption within one month of storage at refrigerator temperature.

Cheeses are more susceptible to spoilage due to microbial contamination. The source of contamination include both intrinsic factors (nutrients, water activity, PH, temperature, inhibiting factors produced by starter cultures and nonstarter microorganisms, (competitive microflora, etc.) and extrinsic factors (microbial quality of raw milk, production phases, ripening and packaging conditions, presence of chemical preservatives), cheese vat, cheese cloth and curd cutting knife, production room air and storage room(Hosni *et al.,*2011).

The improperly cleaned plants, equipment and food handlers are the main sources of contaminations to the cheese. Consumption of contaminated cheese with pathogenic microorganisms and their toxins can be used serious food borne problems to threat public health and big economic losses. But the most traditional cheeses are usually produced under poor hygienic conditions with different manufacturing technologies in local dairy plants.

***Escherichia coli* (*E. coli*) is one of normal inhabitant microorganisms of large intestine in human and warm-blooded animals. The main source of *E. coli* in raw milk and milk products is fecal contamination together with poor hygienic practices (Garbage *et al.*, 2016; Lara *et al.*, 2016). However, certain serotypes of *E. coli* have acquired some virulence-associated genes that enable them to cause intestinal or Extra-intestinal diseases (Hodson *et al.*, 2017).**

E. coli **is generally used as an indicator of direct or indirect fecal contamination and the possible presence of enteric pathogens in milk and dairy products (Kurnai and Johnson, 2001).**

These serotypes that cause enteric infections are generally called diarrheagenic *E. coli* strains, and their pathogenesis is associated with a number of virulence attributes, which vary according to pathotype.

Currently, diarrheagenic *E. coli* strains are classified into seven main pathotypes based on their distinct virulence determinants and pathogenic features, including enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohemorrhagic *E. coli* (EHEC), shiga toxin-producing *E. coli* (STEC), enter invasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAEC) and diffusively adherent *E. coli* (DAEC) , Uropathogenic *E. coli*

Uropathogenic *E. coli* (UPEC) is the most frequent pathogen responsible for up to 80% of UTIs have special virulence factors, including pili or fimbriae, especially type 1 which have the structural rule gene called *Fim H* gene which mediate attachment to uroepithelial and vaginal cells, resistance to human serum bactericidal activity, hemolysins production.

virulence factors of UPEC strains have a significant role in development of UTIs. The most virulence factor dependent upon the UPEC include adhesions type 1 fimbriae encoding by *Fim H* gene (Rutter *et al.*, 2006; Xia *et al.*, 2010) (Zahren et al 2014).

The serotypes associated with illness in humans include members of O serogroups 26, 91, 103, 111, 113, 121, 145 and 157 (Karbala *et al.*, 2010; Pizarro *et al.*, 2014).

As soft cheeses (Kariesh, Talega and Damietta) collected from different localities from Gharbia governorate may subjected to contaminations with *E. coli* and may constitute public hazard.

E. coli **constitutes publics hazards of living creatures especially humans (likes meningoencephalitis, pyelonephritis, hepatitis, pneumonitis, pleurisy, pericarditis, peritonitis, osteomyelitis, appendicitis).**

Therefore, this work was planned to covers the followings points:

1-Isolations, biochemicals and serologically identifications of pathologically pathotypes of Escherichia coli from Kariesh, Tillage and Damietta types of cheeses in el _Gharbia governorate.

2-Molecular identifications of isolated bacteria (Bacterium Coli) using PCR Techniques)

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ON SOME MASTER AND PHILOSPHY DOCTOR THESIS

E COLI CONSIDERED FROM THE FIRST FORMS OF LIFE ON EARTH AND CLASSIFIED ACCORDING TO THE GENOTYPING INTO EIGHT 8 PATHOTYPES :

1-ETEC HAVE THE GENES OF LT AND ST TOXINS MAY THE TWO OR ONE OF THEM
2-EHEC HAVE THE GENE OF HEMOLYSIN
3-EPEC HAVE THE GENEOF INTIMIN GENE EAE A GENE
4-STEC HAVE THE GENES OF SHIGA TOXIN STX1 STX2 GENES
5- EIEC HAVE THE FELAGELLAR GENE
6- EAEC HAVE THE GENE OF K MICROCAPSULE
7-DAEC HAVE THE COMMON AGREGGATING PILLUS GENE
8-UPEC HAVE FIMBERIAL H GENE HAS AFFINITY TO GENITOURINARY TRACT EPITHELIUM

IF YOU CAN NOT MAKE ACONFIRMATIVE MOLECULAR GENOTYPING WE RECOMMENDED TO APPLY SEROLOGICAL IDENTIFICATIONS.
THE MOST IMPORTANT IDENTIFICATIONS OF BIOLOGY IS THE GENOTYPING BECOUSE THE SINGLE SEROTYPE MAY CONTAIN MORE THAN ONE GENE
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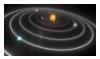

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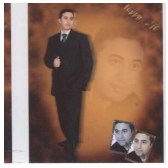

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RECENT DISCUSSIONS MATERIALS FOR RESEARCHER ASSISTANT MOHAMMAD MAHMOUD MOHAMMAD YOUSEF WEHABA 28109091800516-20045788019 66.pdf

WORLD MAP
1. Netherlands
2. Belgium
3. Luxembourg
4. Switzerland
5. Slovenia
6. Croatia
7. Bosnia and Herzegovina
8. Czechia
9. Slovakia
10. Austria
11. Hungary
12. Serbia
13. Moldova
14. North Macedonia
15. Albania
16. Cyprus
17. Lebanon
18. Guinea-Bissau
19. Guinea
20. Ghana
21. Togo
22. Benin
23. Cameroon
24. Equatorial Guinea
25. Rwanda
26. Cambodia
27. Panama
28. Malawi
29. Liechtenstein
30. Montenegro
31. Kosovo
32. Palestinian Territories
33. St. Vincent & the Grenadines
0 2,000 4,000 Miles
0 2,000 4,000 6,000 8,000 Kilometers
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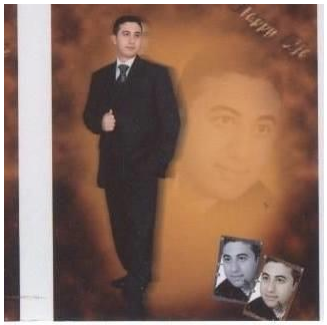
مستندات وشهادات المناقشة العلمية والعملية والدورات والتويفل والتقييم العام من قبل إدارة جامعة بنها العليا للحصول على درجتى الماجستير والدوكتوراة

DISCUSSION STIPULATED DOCUMENTS AND TRAINING QUALIFIED COURSES AND WORKSHOPES AND TOFEL DEGREES CERTIFICATES FOR GAINING MASTER AND DOCTORATE RANKS FOR ALL ESPECIALITIES AND ESPECIALISTS



Salmonella

- Comprise a large group of serotypes (2700)

- World wide & infect a wide variety of hosts

As man , animals & poultry

- Salmonella are typical intestinal pathogens

& contaminate the environment by the organism in the feces and transfer infection to other

Some species are host spcific

species	group	host	disease
S.Paratyphi A	A	Man	Paratyphoid A fever
S.Paratyphi B	B	Man	Paratyphoid B fever
S.Abortus equi		Equine	Equine abortion & infertility
S.Abortus ovis		Ovines	Ovine abortion
S.cholera suis	C	swine	Piglet typhus Infectious enteritis
S.typhi	D	man	Human typhoid fever
S.gallinarum		poultry	Pullorum disease

salmonella

Genus : Salmonella

Species : S. typhi

S. typhimurium

S. enteriditis

S. gallinarum-pullorum

S. dublin

S. infantis

S. derby

S. agona

S. panama

S. heidlberg

morphology

- Cocco-bacilli
- Gram -ve
- Medium sized rods
- Actively motile expect S.gallinarum and pullorum
- Many species developed fimbria
- Non spore forming
- Non capsulated

Cultural characters

- **Faculatative anearobes . Temrpature 37C PH 7.2 incubation time 48 hours**
- **Grow in ordinary media**
- **In nutrient broth** **nutrient agar**

Turbidity , without Pellicle formation

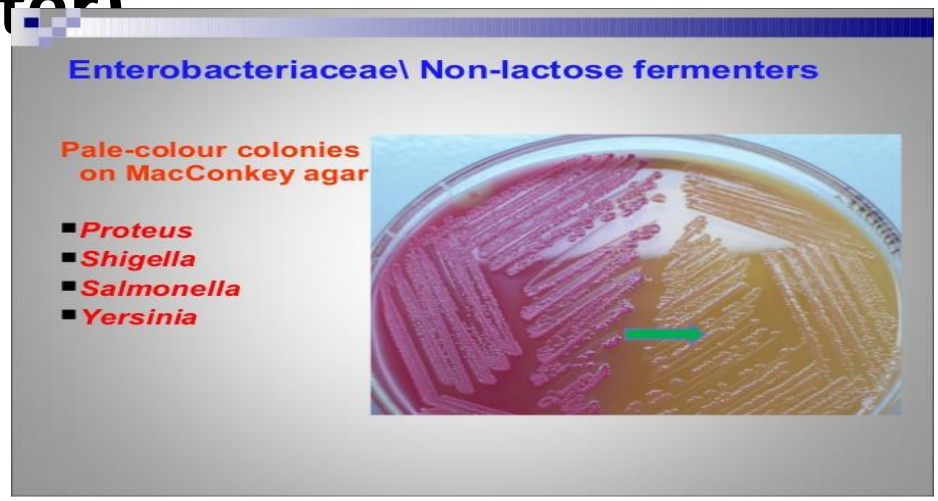
round colonies, smooth, convex with grayish colour

S. gallinarum & S. abortus ovis

small, dew drop like colonies after 48 hours incubation

- **Onto blood agar ⟶ the colonies relatively large , non haemolytic**
- **Onto MacConkey agar**

pale in colour (non lactose fermenter)

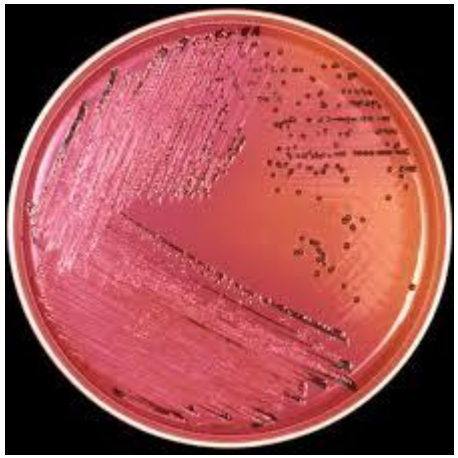


Selective media of Salmonella

There two selective media of salmonella

1- XLD (Xylose Lysin Deoxscholte) agar : colonies appear pink calour with black center due to salmonella produce H2S

2- S.S agar (Salmonella Shigella Agar) : give yellow coloniesn



Enrichment media

There is a more complex problem for salmonella isolation due to their presence in the intestinal tract

Examples :

- Tetrathionate brilliant green broth

- Selenite F broth

by growing the fecal samples or intestinal contents on the enriched media for 18 -24 hours to inhibit the growth of the other contaminants as E.coli , proteus

Biochemical reactions

- Lactose -ve
- Indole -ve
- MR +ve
- VP -ve
- Citrate +ve
- Urease -ve
- H2S +ve

Viability & resistance

- Salmonellae easily killed at 60ºC for 20 min.

- Quickly destroyed by the common chemical disinfectant

- **Identification of salmonella species depends mainly on the serotyping according to**

Kaufman & White scheme of the antigenic structure of salmonella

Antigenic structures

- **Somatic antigens O-antigen**

- **Present in the cell wall**

- **Lipopolysaccharide-protein complex**

- **Designated by arabic numbers from 1 – 65**

- **The majority of salmonallae posses more than one of somatic antigens (3-4) on their surface**

- **Flagellar & fimbrial antigens (H- antigens)**

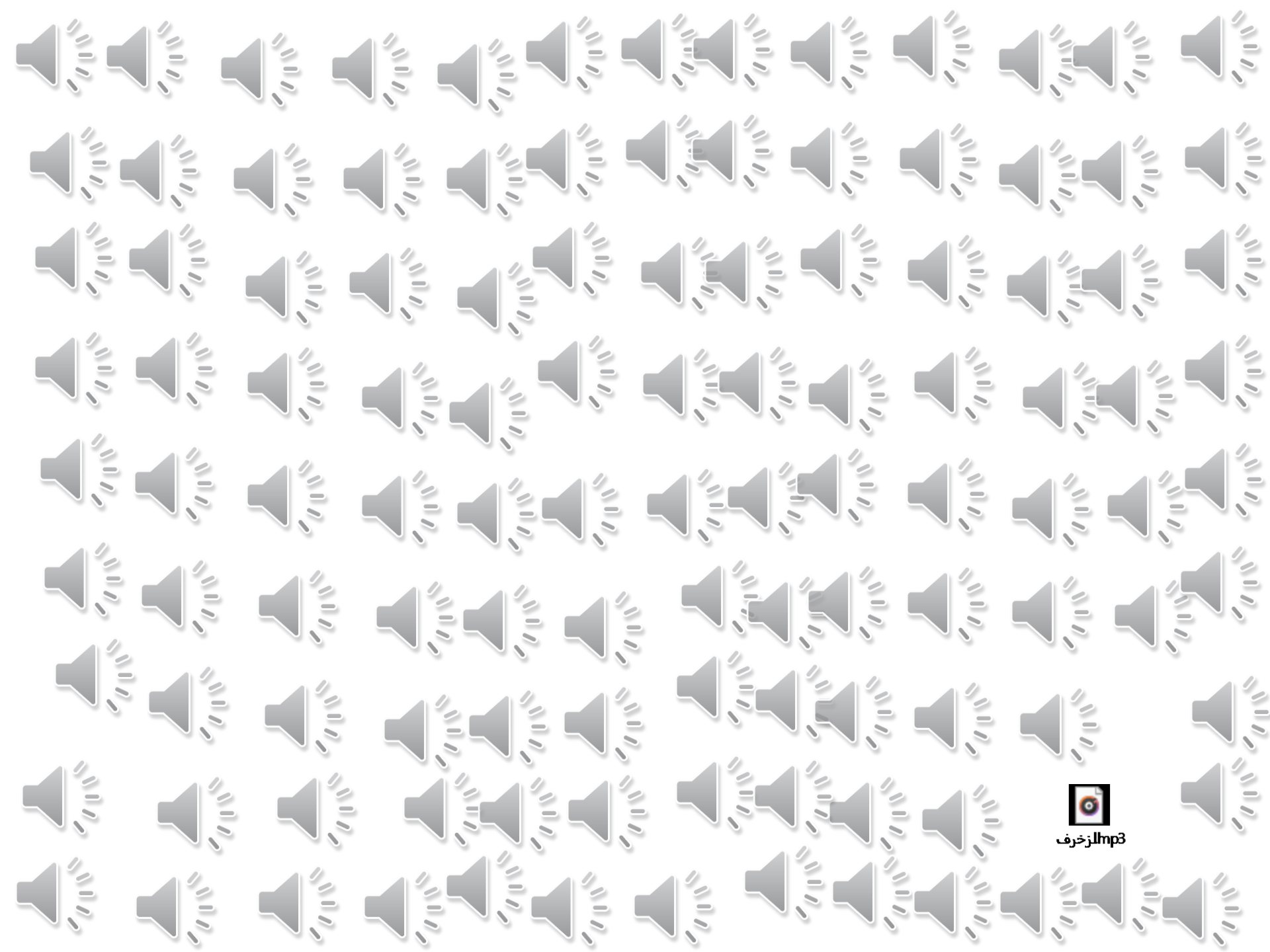
- **Present in motile strains except S.gallinarum**
- **- protein in nature**
- **- salmonella have 2 phases of flagellar antigens**

- **Phase 1 H** — **phase 2 H**
- **Identified by alpha-** — **20 H antigens are**
- **betical a,b,c,d ….** — **Signified by 2 numbers**
- — **1,2 – 1,3 etc**
- — **or 2 -3 letters as I,W , enx**
- — **enz ……**

- **Virulent antigen (Vi – antigen)**
- **Present in freshly isolates of S.typhi**
- **Vi – antigen lost by subculturing**

Examples of salmonella serotypes

	S. paratyphi A	S. paratyphi B
group :	A	B
O-antigen :	1,2,12	1,4,5,12
H-antigen		
phase 1 :	a	b
phase 2 :	-	1,2
	monophasic	Diphasic


الزخرف.mp3